The New England
Journal of Medicine

VOLUME 212 MARCH 14, 1935 NUMBER 11

THE CHEMICAL NATURE OF CATARACT IN THE DIABETIC

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THE rôle that diabetes may play in the production of cataract has been controversial for the past 150 years. The advent of the slit-lamp and corneal microscope two decades ago made possible for the first time an intimate and minute study of the crystalline lens in its normal and abnormal states. The slit-lamp technic has revealed no type of lens degeneration typical of diabetes unless it be the bilateral flocculent or snowflake opacities situated in the lens cortices of juvenile diabetes, a degeneration which is slowly progressive and may be accompanied by opacities that conform to the so-called "Complicated Cataract." Similar floceuli or snowflake opacities have been found by slit-lamp in parathyroid tetany (in which the calcium balance is upset) scleroderma, and myotonic dystrophy.

The morphology of cataracts found in diabetes is fully discussed in the preceding paper by J. Herbert Waite, M.D. and William P. Beetham, M.D.* They found approximately the same cataract frequency in diabetics (11 per cent) as in non-diabetics (10 per cent) and could demonstrate no type of cataract distinctive of diabetes.

The chemistry of the cataractous eye has been of interest to investigators for sixty years. The lipid content of the lens was early detected, but von Graefe[2] was probably the first to publish a report of his discovery of the presence of cholesterol crystals in the human lens. The first quantitative chemical study for lipids was made by Cahn[3] in 1881. His findings are as follows:

	(On basis of dry material)
Normal lenses	0.62 per cent cholesterol
Cataractous lenses	4.55-6.22 per cent cholesterol

His results are evidently computed on the basis of dry lens material. If one recalculates Cahn's figures on the basis of Salit's[4] finding that the water content of lenses is approximately sixty per cent, then Cahn's figures for cholesterol become somewhat lower.

	(On basis of fresh material)
Normal lenses	0.248 per cent cholesterol
Cataractous lenses	1.82-2.48 per cent cholesterol

Salit[5] found the range of the cholesterol content of twenty-nine individual cataractous lenses to be from 0.23 per cent to 0.89 per cent with an average of 0.61 per cent. Two normal human lenses of children had 0.182 per cent and 0.174 per cent respectively and a normal adult lens contained 0.275 per cent cholesterol. The senile cataractous lenses studied by Cahane[6] had an average of 0.62 per cent whereas the cholesterol content of the lenses of three patients who did not have cataract were 0.21, 0.42, 0.42 per cent. Cahane studies also demonstrated that the cholesterol content of the lens of six calves was 0.102 per cent. Bürger and Schlomka[7] using fresh cattle lens material found the cholesterol to vary with age rising from 0.056 per cent to 0.22 per cent.

Calcium is a normal constituent of the human lens. Calcium deposits have been observed in both clear and opaque lenses after the fiftieth year[8], but were not found in younger lenses[9]. Adams[10] found the normal human lens to contain an average of 18.5 mg. calcium although other investigators (Cahn[3], Burge[11], Bürger and Schlomka[7] and Salit[12]) found no calcium or traces only in either animal or human lenses. All observers have found relatively large amounts of calcium in the cataractous eye. The amounts vary widely and average values are as follows:

CALCIUM IN CATARACTOUS LENSES	
(Burge)	12.5 mg. per cent
(Salit)	35.4 mg. per cent
(Adams)	65.3 mg. per cent

Inorganic constituents of the lens have been determined by other observers, but one does not find values for phosphorus. Cahn[3] finds the soluble and insoluble salts of the normal lens to be 1.36 per cent and 0.46 per cent respectively and of the cataractous lens to be 2.1 per cent and 1.2 per cent respectively. Kirby and Wiener[13] state that in the ageing process the "ash of the lens increases from an initial concentra-

*All of the analytical procedures were worked out with extreme care and carried out by Dr. Helen Updegraff Carey, whose sudden death by a totally unexpected embolism at the completion of the analyses necessitated the writing of this paper by the joint author. The technic of analysis required nearly six months' constant work to perfect and when once perfected required careful control through every step. Fortunately the analytical work was completed at the time of Dr. Carey's untimely death and the precise and perfect condition of her notebooks made the task of reporting her data in this paper possible.

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tion of 3 per cent to approximately 3.8 per cent. The increase is mainly in the calcium and phosphorus. The calcium increases 15 per cent of its initial value and the phosphorus 26 per cent." No values are given for the phosphorus content of the cataractous lens. The slit-lamp and corneal microscope detected crystals in the lens of a patient of Braun[14] which appeared like concretions of calcium phosphate. When the lens was extracted and subjected to analysis, no phosphate could be found.

In all the studies made on the cataractous lens very few analyses have been made of the lens of diabetic patients. In the series of forty-four in which Salit[12] determined the calcium content of cataractous lens, two only were diabetic. The calcium content of these lenses from diabetic patients was 19.1 mg. per cent and 19.7 mg. per cent respectively as compared with a range of 2.8-108 and an average for the group of 35.4 mg. per cent. In the chemical studies of lipids in twenty-nine cases of cataract by Salit two patients had diabetes. The cholesterol content of the lenses of these two diabetics was 0.51 per cent and 0.61 per cent as compared with a range of 0.23 per cent-0.89 per cent and an average for the group of 0.61 per cent.

MATERIAL

This report is based on the chemical analysis of fifty-five human lenses of which ten were normal, thirty were cataractous lenses of non-diabetic patients and thirteen were cataractous lenses of diabetic patients, one was a lens with glaucoma and one a dislocated calcified lens. A determination of cholesterol, calcium and non-lipid phosphorus was done on each individual lens in most cases. The lenses were all removed in capsule. The normal lenses were obtained at the autopsy table except in the instance of the dislocated lens. The cataractous lenses were removed by operation at the New England Deaconess Hospital or the Massachusetts Eye and Ear Infirmary. Six flocculi cataracts in juvenile diabetics have been operated, but, because of the minute amount of material removed, no analyses have been made on them.

TECHNIC AND METHODS

The lenses removed in capsule were placed in paraffin lined ground glass covered containers and taken as soon as possible to the laboratory. Here they were quickly weighed, the early ones on a regular Becker analytical balance and the later ones on a Bang micro balance. The weights varied from 0.1384 Gm. to 0.3189 Gm. The lenses were placed immediately in approximately 5 cc. of redistilled 95 per cent alcohol and the containers tightly stoppered. As a rule the time elapsing between extraction and analysis has been from five to six hours. If the operation was performed at the Deaconess Hospital the time interval has been less than one hour. If the container is well stoppered the lens may be left for an indefinite period immersed in alcohol until analyzed. When ready to analyze, the lens material was crumbled in the alcohol with a small glass rod and transferred quantitatively to a small fluted hardened filter paper previously extracted with hot 95 per cent alcohol. When the filter paper was dried it was folded into a small packet containing the lens material and transferred to an extraction apparatus, where it was refluxed with alcohol for twenty-four hours. This time has been found adequate to remove all material giving the Liebermann Burchard reaction. A cholesterol determination using a modification of the Bloor, Pelkan and Allen[15] method was made in duplicate on the combined alcoholic extract. Analysis for calcium and phosphorus was made on the lens material which had been extracted with alcohol. The residue, left in the filter paper after the alcoholic extraction was transferred quantitatively and with extreme care to a small beaker. Here it was treated with concentrated nitric acid and gently warmed. When completely dissolved it was transferred quantita-

TABLE I

NORMAL OR NON-CATARACTOUS LENSES

No.	Source	Weight of Lens in Gm.	Cholesterol mg. %	Calcium mg. %	Phosphorus (Non-Lipid) mg. %	Ca/P Ratio
1.	Autopsy	0.2982	313			
3.	"	0.3144	398	10.0		
6.		0.2617	398	9.2	12.6	0.7
11.		0.3606	282	8.9	8.5	1.0
†19.	"	0.2718	374	11.8		
23.	"	0.2854	476	9.8	19.5	0.5
24.	"	0.2582	510	13.9	17.3	0.8
93.	Dislocated Normal Lens	0.2187	427	12.1	21.6	0.6
*275.	Autopsy	0.2425	516	3.3	18.5	0.2
*289.	"	0.2570	366	6.2	18.4	0.4
10.	Averages	0.2681	406	9.5	16.5	0.6

*Lenses from Patients with Diabetes.
†Other eye of this individual is number 20 with Glaucoma.
(See Table V.)

TABLE II

CATARACTOUS LENSES OF PATIENTS WITHOUT DIABETES

No.	Character of Lens	Weight of Lens in Gm.	Cholesterol mg. %	Calcium mg. %	Phosphorus (Non-Lipid) mg. %	Ca/P Ratio
13.	Mature Cataract	0.2240	349	55.4	26.6	2.1
14.	Mature Cataract	0.2777	412	30.3	7.0	4.3
25.	Cataract (Parathyroid tetany)	0.1669	622	52.0	13.8	3.8
47.	Immature Cataract O. S.	0.3189	527	7.5	20.0	0.4
48.	Mature Cataract O. U. O. D. extracted	0.2931	424	27.3	10.0	2.7
49.	Immature Cataract O. S.	0.2484	451	11.3	30.5	0.3
50.	Immature Cataract O. S.	0.1479	685	32.5	24.0	1.4
51.	Mature Cataract O. S. Sec. Glaucoma O. S.	0.2071	395	73.4	38.1	1.9
52.	Mature Cataract O. S.	0.2018	673	35.7	10.7	3.5
53.	Immature Cataract O. U. O. S. extracted	0.1842	452	21.7	24.8	0.9
54.	Immature Cataract O. U. O. D. extracted	0.1974	382	16.2	22.3	0.7
55.	Mature Cataract O. U. O. D. extracted	0.3079	424	27.3	12.9	2.1
56.	Hypermature Cataract O. S.	0.2185	430	29.3	12.2	2.4
58.	Immature Cataract O. U. O. D. extracted	0.2516	407	22.3	12.9	1.7
59.	Immature Cataract O. U. O. D. extracted	0.2007	502	27.9	16.5	1.6
60.	Immature Cataract O. U. O. D. extracted	0.1710	717	0.0	27.8	0.0
61.	Immature Cataract O. U. O. S. extracted	0.1810	691	61.9	26.9	2.3
62.	Immature Cataract O. U. O. S. extracted	0.2222	572	3.6	25.4	0.1
63.	Immature Cataract O. U. O. S. extracted	0.2138	619	29.9	21.5	1.4
71.	Mature Cataract	0.1690	635	51.1	13.4	3.8
89.	Nuclear Cataract O. S.	0.2548	463	3.3	19.5	0.2

TABLE II (*Concluded*)

No.	Character of Lens	Weight of Lens in Gm.	Cholesterol mg. %	Calcium mg. %	Phosphorus (Non-Lipid) mg. %	Ca/P Ratio
189.	Immature Cataract	0.2001	521	35.0	17.2	2.0
192.	Mature Cataract	0.1824	523	42.8	22.6	1.9
193.	Mature Cataract	0.2349	447	26.4	17.0	1.6
195.	Mature Cataract O. S.	0.1384	531	47.4	15.0	3.2
196.	Mature Cataract O. S.	0.1289	622	17.1	18.6	0.9
197.	Mature Cataract	0.1797	519	47.9	12.0	3.9
201.	Hypermature Cataract O. S.	0.1942	601	46.3	15.4	3.0
202.	Mature Cataract O. S.	0.1843	622	7.6	29.3	0.3
203.	Immature Cataract	0.2145	520	8.4	28.9	0.3
30.	Averages	0.2105	525	30.0	19.7	1.8

TABLE III

CATARACTOUS LENSES OF PATIENTS WITH DIABETES

No.	Character of Lens	Weight of Lens in Gm.	Cholesterol mg. %	Calcium mg. %	Phosphorus (Non-Lipid) mg. %	Ca/P Ratio
7.	Mature Cataract O. S.	0.2259	359	33.6	4.2	30.5
8.	Hypermature Cataract O. D.	0.2967	456	26.7	3.9	6.8
57.	Cataract	0.1797	443	57.9	3.2	18.0
100.	Mature Cataract O. S.	0.1856	656	46.8	14.3	3.3
125.	Immature Cataract O. S.	0.1690	528	2.4	7.5	.3
211.	Cataract O. D.	0.1856	510	23.7	10.0	2.3
219.	Immature Cataract	0.1790	587	24.6	4.4	5.6
235.	Complicated	0.1815	547	30.9	0.6	51.5
236.	Cataract O. S.	0.1700	552	56.5	1.1	51.4
251.	Cataract O. D.	0.1945	487	37.0	2.3	16.1
286.	Mature Cataract O. D.	0.2145	589	61.5	7.7	8.0
290.	Mature Cataract O. D.	0.2570	386	37.4	5.8	6.5
293.	Cataract O. S.	0.1715	552	16.3	15.7	1.0
13.	Averages	0.2008	511	35.0	6.2	16.0

tively to a digestion tube, evaporated to loss of nitric acid and digested with H_2SO_4 to a water-clear solution. This is a long and tedious process especially if the lens contains much organic material. The calcium was precipitated in the digestion tube, filtered through a Fiske filter tube and analyzed by the Fiske-Logan Method[16] for calcium.

The filtrate from the calcium determination was freed from ammonium oxalate (by digestion with sulphuric acid) and its phosphate content determined by a modification of the Fiske-Subbarow Method[17] for phosphorus.

Throughout the whole procedure blanks were run on all reagents used in every determination and with every set of four tests controls and blanks were run.

RESULTS

In the normal or non-cataractous lens the cholesterol values varied from 282 to 510 mg. per cent, the average value being 406, a figure approximately twice the normal value for cholesterol in the blood. Calcium was found in every one of the non-cataractous lenses examined. The values for calcium in these lenses are approximately the same as normal blood calcium values, 9.5 mg. per cent, although the range is slightly wider. The average value for phosphorus in the normal lens determined by the method described is 16.5 mg. per cent, a value approximately four times the phosphorus of normal blood plasma. It is interesting to note that the lens calcium of the two patients with diabetes is very low in each instance, whereas the phosphorus figure is slightly higher than the average in these two instances. The Ca/P ratio for the clear normal lenses is 0.6. In all of the tables the Ca/P ratio is calculated on weight in mg.

All the cataractous lenses showed slightly higher values for cholesterol content than the normal lenses, the average cholesterol value for the cataractous lens of the non-diabetic being 525 mg. per cent and the cataractous lens of the diabetic 511 mg. per cent. The calcium values in both groups of cataractous lenses were definitely elevated, the cataractous lens of the non-diabetic having an average of 30.0 mg. per cent calcium and cataractous lens of the diabetic having an average of 35.0 mg. per cent as against 9.5 mg. per cent in the normal lens.

A marked difference was found between the two groups of cataractous lenses in respect to their content of phosphorus. The average value for phosphorus in the cataractous lens of the non-diabetic was found to be 19.7 mg. per cent and in the cataractous lens of the diabetic 6.2 mg. per cent as against 16.5 mg. per cent in the non-cataractous lens. The average Ca/P ratio in the cataractous lens of the non-diabetic is 1.8, whereas in the cataractous diabetic eye the Ca/P ratio is 16.0. If one calculates the Ca/P ratio of $Ca_3(PO_4)_2$ on the basis of weight it is 1.9. This figure appears significant when compared with the Ca/P ratio found in cataractous lenses, but whether all the phosphorus of the lens tissue is inorganic is debatable.

TABLE IV

CHEMICAL COMPOSITION OF CATARACTOUS AND NON-CATARACTOUS HUMAN LENSES

	No.	Cholesterol mg. %	Calcium mg. %	Phosphorus (Non-Lipid) mg. %	Ca/P Ratio
Normal Lenses	10.	406	9.5	16.5	0.6
Cataractous Lenses					
A. Non-diabetic	30.	525	30.0	19.7	1.8
B. Diabetic	13.	511	35.0	6.2	15.5

TABLE V

CHEMICAL COMPOSITION OF LENSES HAVING ABNORMALITIES OTHER THAN CATARACT

No.	Character of Lens	Weight of Lens in Gm.	Cholesterol mg. %	Calcium mg. %	Phosphorus (Non-Lipid) mg. %	Ca/P Ratio
16.	Dislocated and Calcified Lens	0.1046	402	14072.0	6348.0	2.2
20.	Glaucoma	0.2788	353	201.0		

The normal lens has a cholesterol content approximately twice the value of cholesterol in normal blood. The cataractous lens has a cholesterol value approximately two and one-half times higher than the normal blood cholesterol. There is no difference in cholesterol content between the cataractous lens of the diabetic and the non-diabetic individual. The normal lens has a calcium value approximately the same as blood and this value is markedly increased in the cataractous lens. The normal lens has a value of 16.5 mg. per cent phosphorus (non-lipid), the

cataractous lens of the non-diabetic has a phosphorus content slightly higher. The cataractous lens of the diabetic has a markedly reduced phosphorus content.

The following values for chemical constituents of the two lenses having abnormalities other than cataract are included in this paper. They are of no special interest in this report, but we are using this means of making the data available to the other workers who may be interested in studies of abnormal lenses.

SUMMARY

(1) Reports are given on chemical analyses for cholesterol, total calcium and total non-lipid phosphorus of ten normal lenses, thirteen cataractous lenses of diabetic patients, thirty cataractous lenses of non-diabetic individuals and two unusual abnormal lenses.

(2) The normal lens contains approximately 400 mg. per cent cholesterol, 9.5 mg. per cent calcium, 16.5 mg. per cent phosphorus.

(3) The cataractous lens contains 522 mg. per cent cholesterol, or approximately 20 per cent more than the normal lens.

(4) The calcium content of the cataractous lens is 31.6 mg. per cent, a value three times the value in normal lens material or in normal blood.

(5) The phosphorus (non-lipid) found in the normal lens is 16.5 mg. per cent and the calcium phosphorus ratio is 0.6 per cent. In the cataractous lens of the non-diabetic the average phosphorus value is 19.7 mg. per cent, a value higher than in the normal lens, and the calcium phosphorus ratio is proportionately higher. In the cataract of the diabetic the phosphorus value is strikingly lower than in the cataract of the normal lens or of the lens of the non-diabetic individual. The average value is 6.2 mg. per cent. The calcium phosphorus ratio is correspondingly high, 16.0 per cent.

The cataract forming in the eye of the diabetic appears chemically to be the classical senile cataract in all respects save one. The phosphorus metabolism is markedly different. What importance this has one does not know at present.

This study carried on over a period of three years was made possible by a grant from Mr. and Mrs. Francis P. Garvan and the Chemical Foundation.

The writer wishes to acknowledge the kind coöperation of Dr. Elliott P. Joslin and Dr. J. Herbert Waite throughout this study. I am grateful also to the helpful suggestions made by Dr. Y. Subbarow and Dr. M. A. Logan.

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CHRONIC GASTRITIS*

A Clinical Discussion Based on Gastroscopic Examination

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ACCORDING to Osler[1] "the term chronic gastritis is used loosely to designate a variety of gastric disorders, in many of which there are no actual changes in the mucous membrane. If the term is restricted to organic changes, it is a comparatively rare primary disease. Clinically it is not easy to draw the line between chronic gastritis and functional disturbance." More recently, Thomas R. Brown[2] writing in Cecil's Text-Book of Medicine states that "only when the symptoms seem to be evidence of persistent chronic inflammatory changes in the stomach should the term 'chronic gastritis' be employed." Up to the present time, then, it is seen that the term has been vaguely employed and the diagnosis far from exact.

Few of us, however, will deny the existence of chronic gastritis. Stomatitis, esophagitis, duodenitis, enteritis, appendicitis, colitis, and proctitis are all terms in generally accepted use to-day. The term gastritis, however, because of its loose meaning in the past, has fallen into disrepute. The development of the Wolf-Schindler flexible

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gastroscope has now placed at our disposal an easy and positive means for making an accurate diagnosis of gastritis, and the term is at last finding its rightful usage. For a general discussion of gastroscopy with the flexible instrument reference is made to Schindler[3], Henning[4], and Benedict[5].

The etiology of chronic gastritis is not known, but dietary indiscretions, rapid and irregular habits of eating, improper mastication, and excessive use of tobacco and alcohol are undoubtedly of importance. Chemical and bacterial factors may also play a part. Psychogenic instability, which seems to be significant in peptic ulcer, probably also plays an important rôle in gastritis.

Chronic gastritis presents both gross and microscopic pathology. Gross changes consist in edema and reddening of the mucosa, excessive secretion of mucus, hypertrophy of the rugae, granular, verrucous (état mamelonné), or polypoid irregularity of the mucosa, areas of submucous hemorrhage, and mucous membrane hemorrhages with or without visible erosions. Other types of chronic gastritis show a thin atrophic mucosa. Microscopically there may be round cell infiltration, glandular atrophy, goblet cell metaplasia, cystic enlargement of gland remnants, and proliferative changes in the mucosa. Variations in the amount of connective tissue proliferation in different areas will lead to thickening or thinning of the mucosa.

The symptoms are notoriously vague. Distress, pain, nausea, vomiting, eructations, heartburn, gas, anorexia, and fulness may all be present in varying degrees, or largely absent. There is usually something, however, to direct attention to the stomach; and the symptoms are occasionally very suggestive of peptic ulcer, with relief of pain by food or soda. In other words, as Henning says, "so-called ulcer complaints are in no way pathognomonic of ulcer." In my experience epigastric pain or distress has been the most constant complaint; gas, nausea, and vomiting are also of frequent occurrence. Hematemesis and melena are not uncommon. As to physical findings, the teeth are usually poor, the tongue is often coated, and there is frequently some tenderness in the epigastrium. Gastric analysis is said by some authors to show a low acidity in most cases but both normal and hyperacidity have occurred frequently in this clinic.

The diagnosis of chronic gastritis has rested heretofore on insecure foundations, having been based largely on the rather indefinite symptomatology and the exclusion of other organic pathology by x-ray. With the introduction of the flexible gastroscope, however, we now have an easy and accurate diagnostic method at our disposal, the examiner being able to inspect the gastric mucosa with great care and note varia-

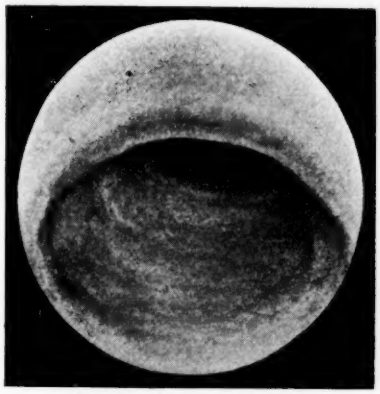

FIG. 1. Normal pylorus.

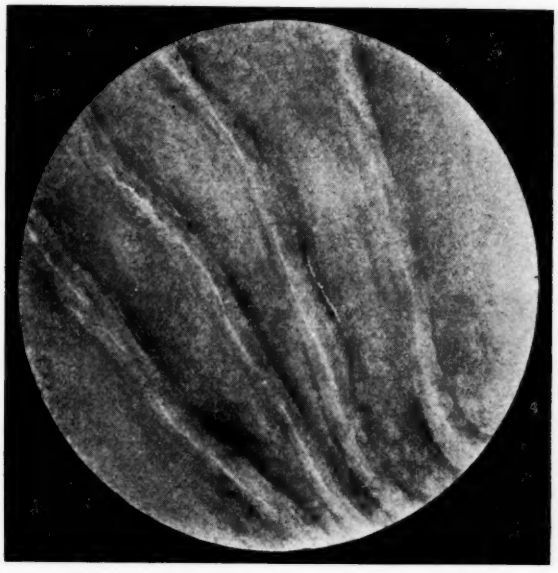

FIG. 2. Normal thin parallel rugae of anterior wall and lesser curvature.

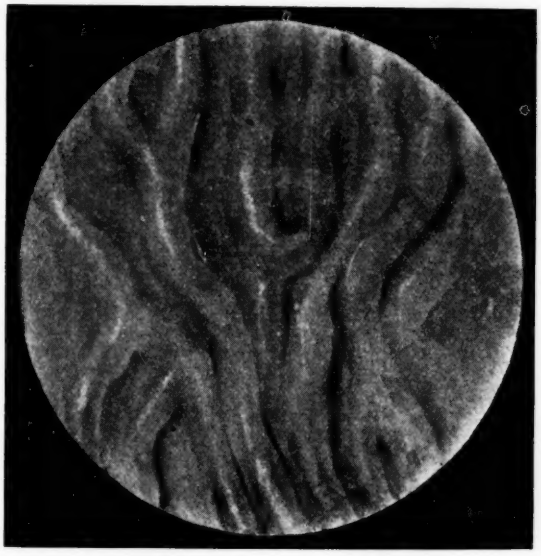

FIG. 3. Normal thick rugae of posterior wall and greater curvature.

FIG. 4. Hypertrophic gastritis.

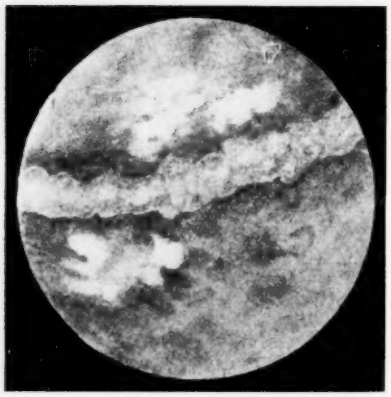

FIG. 5. Verrucous gastritis. Irregular granular mucosa with warty elevations.

FIG. 6. Erosive gastritis. Large erosion in centre of hypertrophied fold.

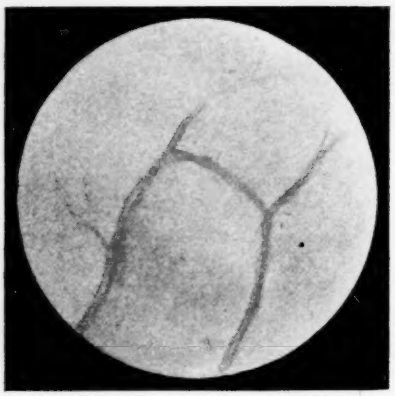

FIG. 7. Atrophic appearance seen especially in advanced pernicious anemia. Note the pale mucosa through which small blood vessels are plainly visible.

tions from the normal (figs. 1, 2, 3*). These may consist in hypertrophic (fig. 4), verrucous (fig. 5), erosive (fig. 6), or atrophic (fig. 7) changes, but, as different types may occur in varying degrees in the same stomach, a definite classification of chronic gastritis according to these terms is not possible in all cases. One may frequently note, however, that the predominant characteristic is hypertrophy or atrophy with or without erosions. The color and character of the mucosa are of very great importance, the color in chronic inflammation being a more intense red than normal, and the mucosa often presenting a very glistening appearance. Glairy, tenacious mucus may be prominent on the surfaces. The rugae may be enlarged and tortuous, with reddening of their crests. Not infrequently the mucosa between the folds presents a granular or warty appearance, which, if predominant, justifies the term verrucous gastritis. When these nodular formations are particularly marked, a polypoid appearance is sometimes presented. These pseudopolyps have occasionally been so large as to have caused an erroneous x-ray diagnosis of polyposis of the stomach. Atrophic changes in the mucosa are also easy to recognize; the normal folds are partially or completely flattened out, so the mucosa presents an unusually smooth surface. When inflamed, there is marked reddening and often glistening of the mucous membrane, but in advanced atrophy and anemia, as for example in pernicious anemia, the surface is very pale and thin, and against the pale background the arterioles stand out very clearly. In the normal stomach and in hypertrophic gastritis, owing probably to the thickness of the mucosa, blood vessels are not visible. Erosions may occur with atrophic or hypertrophic changes; they are small surface defects in the mucous membrane which may or may not be actively bleeding at the time of observation. Bright red areas of irregular size and shape may be seen in some cases just beneath the mucosa. These have been described as evidence of mucous membrane catarrh, and may be due to changes in blood distribution or to submucous hemorrhage.

Very recently roentgenologists have been attempting the diagnosis of hypertrophic gastritis by the so-called "relief" method. By this method small amounts of barium are used, the abdomen is compressed, and an outline of the folds stands out in relief. Thus it may be possible to judge the size and course of the rugae, and when they are large and tortuous the roentgen diagnosis of hypertrophic gastritis is suggested. Gastroscopic confirmation is necessary, however, as there is no substitute for direct examination of the mucosa. I have seen cases where the rugae appeared hypertrophic and

*Photographs taken from gastroscopic drawings made by Miss Muriel McLatchie. Photography through the gastroscope is possible only when a rigid instrument is used.

tortuous by x-ray, and yet on gastroscopy, with moderate inflation of the stomach, the rugae largely disappeared and the mucosa appeared normal. In fact, hypertrophic rugae have been described by x-ray when gastroscopy revealed a definitely atrophic mucosa. The granular appearance so characteristic of verrucous gastritis, and so easily seen by gastroscopy, is entirely missed by x-ray examination. Small erosions also escape diagnosis by x-ray. The two methods are of course supplementary, the x-ray being better, generally speaking, in large tumors and ulcers, and the gastroscope being superior in gastritis. Henning says, "Gastroscopy presents by far the most profitable method of examination in the diagnosis of gastritis."

Chronic gastritis may occur with gastric ulcer, duodenal ulcer, and gastric carcinoma, but perhaps of greater interest and significance is its occurrence alone as a definite disease entity. During the past year and a half a diagnosis of chronic gastritis as an accompanying lesion has been made by gastroscopy in many cases of peptic ulcer and gastric carcinoma. To date about 200 gastroscopic examinations have been conducted in this hospital, many of them for ulcer and cancer, but in forty patients, in whom no ulcer or neoplasm was demonstrable by x-ray, a diagnosis of chronic gastritis was made by gastroscopy.

No case report can be typical of chronic gastritis, as the picture is so variable. The following report, however, is presented as fairly characteristic of the disease:

J. De C., M. G. H. No. 316919, male, aged thirty-nine, entered the hospital complaining of gnawing epigastric pain of three years' duration. The pain was non-radiating, not very severe, unrelated to meals, and unrelieved by food. Soda had not been tried. For the past two years the pain had been periodic, coming on about every one to two months, associated with anorexia, nausea and vomiting. During the two year period there had been a loss of about twenty-five pounds in weight. The patient was extremely nervous, smoked one and a half to two packages of cigarettes a day, ate very irregularly, and slept only five hours at night. There was no alcoholic history. Physical examination showed a well-developed and nourished individual, with several teeth missing, definite pyorrhea, and some deep tenderness in the mid-epigastrium. Gastric analysis was not done after a regular test meal, but a vomited specimen showed a total acidity equivalent to 40 cc. N/10 NaOH. Gastrointestinal x-ray was done three times, and showed spasm of the antrum of the stomach with slight thickening and distortion of the gastric rugae; the examination was considered negative. On gastroscopic examination, however, the rugae appeared smaller than usual but were irregular in their course; the mucosa was definitely granular, justifying a diagnosis of verrucous gastritis. The treatment in this case consisted in a bland diet, belladonna, dental attention, and general instructions regarding regular habits of eating, sleeping, defecation, exercise, etc.

In general, the treatment of chronic gastritis should be preventive and curative. Elimination of alcohol and nicotine, and regulation of the patient's daily life will usually accomplish a great deal. A bland diet with frequent feedings at regular hours is of very great importance. Adequate rest and sleep, and freedom from excessive mental or physical strain are also very important. Dental attention can hardly be overemphasized, as it is of the utmost significance, not only from the mechanical point of view of proper mastication, but also from the point of view of focal infection. The tonsils and other foci of infection should also be treated. In severe cases, bed rest and gastric lavage may be necessary. Dilute hydrochloric acid or alkali therapy may be helpful in appropriate cases. Surgery, in the form of extensive gastric resection, has occasionally been performed for gastritis in Germany, but so far has not been considered necessary in this clinic. Operation may be justifiable in gastritis with pyloric stenosis, or when there is suspicion of malignancy. One must not be content to rest on the diagnosis of gastritis if there is any evidence pointing toward malignant disease. In this regard the following history is pertinent:

M. K., M. G. H. No. 337960, male, aged forty-five, first came to the hospital on March 15, 1934, complaining of stomach trouble of three months' duration. The complaints were dull pain occurring an hour and a half after eating, pain at night, anorexia, constipation, and loss of ten pounds in weight. Carcinoma of the stomach was suspected, but x-ray examination showed only enlarged rugae, and a diagnosis of hypertrophic gastritis was made. Gastroscopy two weeks later showed along the greater curvature and posterior wall "many large elevations and depressions, giving the mucous membrane a warty appearance which was so marked in some places as to suggest malignant disease." The gastroscopic examination was not conclusive, however, and more faith was placed in the x-ray diagnosis. Moreover, there was some clinical improvement on dietary management. Two months later, however, because of persistent symptoms and an x-ray somewhat suggestive of malignant infiltration, exploratory operation was done, and an inoperable carcinoma of the stomach was revealed, adherent to the pancreas and infiltrating the gastrohepatic omentum. A large gland in the gastrocolic omentum was removed for biopsy, and showed metastatic colloid adenocarcinoma, grade III.

Comment: Any suspicion of carcinoma of the stomach by repeated x-ray examination has always justified exploratory laparotomy. After this experience exploratory operation should also be indicated when malignant disease is suspected on gastroscopic examination.

As a complication of chronic gastritis, hemorrhage is of very great importance. Gross hemorrhage in this disease has been observed by many writers. To quote again from Henning: "Although in the course of time a long series of observations has been assembled on the occurrence of gross bleeding in gastritis without ulcer, remarkably few clinics are aware of this fact. . . . Konjetzny and Puhl, on the basis

of their very careful histological studies, have come to the conclusion that perhaps in all cases of so-called parenchymatous gastric bleeding a gastritis is present as the essential cause. . . . The author believes that gross gastric bleeding in gastritis is equal in frequency to bleeding from tumors and ulcers." Korbsch[6], too, states "it is certain that gross bleeding can occur in chronic gastritis, and that hematemesis is no longer to be considered a sure sign of ulcer." During the past year at this hospital nearly one half the cases of chronic gastritis have given a history of bleeding. In these cases there has been definite hematemesis or melena, with red counts varying from two to five million, and negative x-rays of the esophagus, stomach, duodenum, and colon. Gastroscopy has shown varying degrees of gastritis in all cases, with erosions and actively bleeding areas in some. As an example of severe bleeding apparently occurring in chronic gastritis the following case is interesting:

A. P. B., M. G. H. No. 332336, a fifty-six year old single white American housekeeper, first entered the hospital September 29, 1933, complaining of dizziness and weakness of four days' duration. For one year she had had intermittent attacks of indigestion, consisting of cramplike epigastric pains, coming on shortly after eating and relieved by vomiting. There was no history of relief of pain by food or soda. Four days before admission there had been gross hematemesis and melena, with fainting. The red blood count on entry was found to be only 1.89 million. Improvement on the ward was rapid, and three weeks later a gastrointestinal series was done which showed hypertrophic rugae but no ulcer. Barium enema was negative. Gastroscopy, however, showed thickened, inflamed rugae with several erosions which could easily have been the source of the hemorrhage. A diagnosis of hypertrophic gastritis was made.

Second admission: About a year later (September 22, 1934), after dietary indiscretion, the patient again had an episode of bleeding and entered the hospital. The red blood count was 2.1 million. Gastrointestinal series now showed a duodenal ulcer. By gastroscopy gastritis was still present, though less marked than before.

Comment: This history records: (1) Gross hemorrhage from the upper gastrointestinal tract where, on the first admission, no demonstrable explanation could be found other than a chronic gastritis. (2) The development of a duodenal ulcer in a patient known to be suffering from a chronic gastritis. The question of the relationship of chronic gastritis to peptic ulcer is not yet solved, but it may be of significance in this case that the chronic gastritis apparently preceded the ulcer. Henning, although at first skeptical, states he has now observed a typical surface ulcer of the stomach develop from a small erosion in the course of four to six weeks; and Konjetzny[7, 8] not only regards chronic gastritis as leading to ulcer but also believes that chronic gastritis may lead to carcinoma. Schindler*, on the other hand, is inclined to regard the gastritis in such cases as secondary.

The prognosis in chronic gastritis must be guarded. As in peptic ulcer, there are likely to be remissions and relapses, though many patients are clinically entirely relieved after a short period of treatment. Such clinical improvement, according to Henning, is in striking contrast to the pathological changes which may still be evident on gastroscopic examination. In general, minor surface changes in the mucosa such as erosions, small areas of hemorrhage, and hyperemia, will heal completely without a trace, but "hypertrophic formations, such as granulations, verrucous humps, and pseudopolyps remain completely refractory." When, on the other hand, diffuse atrophy of the mucous membrane is present, there is probably little hope of regeneration, though in pernicious anemia in this clinic we have had some recent evidence of an improvement in the gastroscopic picture after intensive liver therapy. Henning believes that the ultimate prognosis in chronic gastritis will be based more and more on the pathological picture as seen gastroscopically.

In conclusion the following points should be emphasized:

Chronic gastritis is a definite disease, in the accurate diagnosis of which gastroscopy is the most valuable method of examination.

The importance of chronic gastritis lies not only in its existence as an independent disease, but also in its possible relationship to gastric ulcer and carcinoma.

Hemorrhage is a more common complication of gastritis than is generally supposed.

The development of the Wolf-Schindler flexible gastroscope has provided a safe and practical method for clinical and research use in this field.

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*Personal communication. Professor Schindler is now doing gastroscopy at the Billings Hospital, University of Chicago.

NEW HAMPSHIRE MEDICAL SOCIETY

SOME EXPERIENCES IN THE TREATMENT OF YOUNG DIABETICS*

From the Point of View of the General Practitioner

BY BARBARA BEATTIE, M.D.†

"THE living diabetic child is the most outstanding medical accomplishment of the past decade." This magnificent sentence, the first in Priscilla White's book on diabetes in childhood and adolescence, is full of hope for the future and for me suggests the contrasts between the present era and my first two and a half years in medical school before the discovery of insulin. I will never forget the picture, stamped indelibly on my young mind, of those pre-insulin children, starving to death before our very eyes, in spite of the most expert scientific care and heroic deprivations, poor little stunted bodies with peaked faces and haunted eyes. The doctor, in those days, had to take from his diabetic children the very stuff life is made of and could offer them in its place only a meager existence for a few years, deprived of growth, strength, and all the carefree happiness associated with childhood. The average span of life for the pre-insulin child was two years!

And then the miracle of the ages for the diabetic happened! Insulin was discovered. Under the skins of those doomed children went the first doses of the life-restoring substance; they were allowed to satisfy their hunger again, at first cautiously, for here was a potent preparation not understood very well by anyone in those days, but soon they were eating enough to make them grow and to restore the bloom of childhood to their faces.

To-day we have the diabetic child with us. We have proved that he can grow and develop normally and with but few exceptions, lead a life as full and varied as his non-diabetic playmates. I say we have him with us. As Joslin points out, we are going to have him in ever-increasing numbers, now that we know how to keep him alive. Every year adds another thousand to his group and every general practitioner practicing in a district where there are no specialists, will be called upon more and more to treat the diabetic child. It behooves him to become acquainted with some of the principles underlying the treatment, so that he can at least be an intelligent assistant to the specialist in the nearby city and can certainly instigate treatment for coma while waiting for the specialist's arrival. The principles involved are so few, so simple and so almost mathematical in their precision, that any doctor with a little study can master them and with them, master the disease.

For this reason I have chosen to write down some of my experiences with young diabetics, in the hope that they may be helpful to others with similar problems. I am far from a specialist in diabetes, though I have had some post-graduate work in it and read everything available on the subject. But perhaps the fact that I am not a specialist and know the limitations put upon me by lack of expert knowledge and isolation from easy access to such knowledge, makes my experience of more value to the person in a position like my own.

The young diabetic has the characteristics of the adult suffering from the same disease and certain others peculiar to himself. He begins with the usual poly-triad: polydipsia, polyphagia and polyuria, with their accompaniment of loss of strength and loss of weight. Much more often than his older fellow-sufferer, he plunges into his disease suddenly and it ravages his young growing body in a more spectacular way. His diabetes often seems acute in its early manifestations and it used to be a surprise to me that it could be so easily controlled with comparatively small doses of insulin.

Diabetes in children has, I think, more pronounced nervous symptoms than in adults. I have learned and now teach parents, to watch for irritability, restless sleep, explosive tantrums, as signs of breaking diet. One patient of mine, who often went on a sugar spree, would invariably get so unpleasant around his home the next day that his mother, instead of scolding him for his bad temper, would wisely send him down to me for a scolding of a different sort.

In my experience, diabetes in the young is always progressive during the first few years. It gets worse rather than better until the child has had it for some time, when it appears to become stabilized. I have one patient, now nineteen years old, who has had the disease eight years. Until two years ago she had required gradually increasing amounts of insulin to take care of a maintenance diet. To be sure she was an inveterate offender against her diet and never gave herself a chance to build up her sugar tolerance. Now she seems to have steadied

*A Prize Essay. Under the terms of bequests of Drs. Thomas J. W. Pray and Hosea H. Burnham, the New Hampshire Medical Society awards prizes to its members for approved original essays upon some medical topic. This essay by Dr. Beattie won the prize for 1934.

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down to a constant dose, perhaps because the progress of her disease is arrested, perhaps because she is through with those trying adolescent years and perhaps because she has acquired some sense about taking care of herself. Most likely the reason can be found in a combination of all three.

But even with the children who are conscientious to a pitiful degree about their diets, the disease is still progressive. My most model patient got along beautifully for two years on twenty-five units of insulin a day, kept himself sugar-free and unlike most patients of his age, never had an insulin reaction. He was thirteen when he developed the disease. Now he is fifteen, maturing very fast, growing tall with a sudden spurt and within the past three months has had to be entirely readjusted to diet and insulin, the dose of which has increased to forty-five units. I know he does not break his diet because I put him to bed under close supervision for a week to rule out that possibility. He has not had any acute infection to aggravate his disease. Yet with no apparent cause, except the natural growing-up process, his diabetes has become worse. His blood sugar is more unstable, he spills sugar one day and has an insulin reaction the next. He illustrates very well the striking and spectacular change which comes over these children when they grow into adolescence. When I first began to treat diabetes, I could not believe it possible that the mere changes of puberty could account for such an aggravation of the disease. Now I know that they can. I think this point is not sufficiently emphasized in the literature.

As a matter of fact, instability of the blood sugar is the rule rather than the exception in young diabetics. It is one of the factors which makes them difficult to control satisfactorily. I always attempt to keep them sugar-free but find in actual practice that it is often impossible to keep them both sugar-free and reaction-free for the whole twenty-four hours. A fifteen year old patient of mine illustrates that point graphically. She has never, to my knowledge, been sugar free for any consecutive twenty-four hours and yet for a time she was having insulin reactions at eleven in the morning and again in the middle of the night. She has been a problem to adjust and long ago I gave up trying to keep her urine clear. Once, after a week of indulging in sweets, she nearly went into coma and had a blood sugar of three hundred. After six hours of intensive treatment, she went into insulin shock and her blood sugar was under fifty, though insulin had been given cautiously in small doses every hour and frequent urine tests always contained sugar. In her case, I have felt it more important to control her reactions, than to keep her sugar-free, because I think frequent insulin reactions are fraught with more danger than a mild glycosuria. And furthermore, I believe that it is bad psychology for the young diabetic to get into the habit of nibbling sugar to overcome threatened reactions. It keeps a "sweet tooth" constantly stimulated and before anyone is aware of it, candy is surreptitiously substituted for the sugar. All my diabetics are taught to carry a lump of sugar with them at all times, but never to take it unless they have symptoms of hypoglycemia. It always pleases me, when I ask them about the sugar, to have them produce from the depths of a pocket a dirty crumbly lump which gives mute evidence of disuse!

The adolescent diabetic shares all the characteristics of his younger fellows but adds to them the instability and rebelliousness which go with his age. The "trying teens" are difficult enough in healthy children. But add to the usual ups and downs of adolescence the necessity of an irksome routine, which diabetes inflicts, and you have a situation which often makes scientific management practically impossible. These children are at the age when they resent discipline, rebel against restrictions and feel that they are perfectly able to run their own affairs. Yet the truth of the matter is that they have not yet learned self-discipline and thus have nothing to substitute for the home control from which they insist upon escaping. So far as their diabetes is concerned, they decide early to lead a short life and a merry one, though they know perfectly well that eventually they will pay for it. It has been amazing to me how tenaciously they hang on to this point of view, often going through several coma crises without appearing to learn anything from the experience. When I first began to treat diabetic children these adolescents nearly drove me out of my mind. I couldn't believe that they could be so stupid, so shortsighted and I felt that I must in some way be responsible for their lack of self-control. But now I have grown more philosophical about them and have learned that I must expect frequent dietary indiscretions.

Several years ago I learned a valuable lesson from Joslin. I sent an adolescent boy, who had successfully resisted all my efforts to treat him, down to Joslin with a letter describing the boy's diabetic indulgences in very graphic terms and ending with a plea to put some sense into his head before sending him home. I was amazed at the calmness of Joslin's reply, which said his diet was adequate, suggested a change in the insulin distribution and ended with a casual remark that it was too bad the boy didn't follow his diet more closely! I thought that letter over very carefully and realized once and for all that the boy's doctor could do just so much for him and after that it was all on his own shoulders. These children must learn to take care of themselves, in spite of the dangers they run in doing so and it certainly is a

bad thing to fuss over them too much. There is always the comforting thought to fall back on that if they do get into trouble, the pain and discomfort of acidosis will bring a humble call for the doctor. One of my patients, who was in open rebellion against the whole diabetic situation (with special emphasis on *me*) got herself into impending coma before she admitted she was ill and by the time I got to the house she was screaming to me that if I'd help her get her breath she would never break her diet again. I am sorry to report that she didn't keep her part of the bargain very long that time. She is now three years older and has apparently gained in wisdom, for she has kept out of mischief for a longer period of time than ever before since she developed the disease.

In the treatment of young diabetics I have certain general principles which I adhere to, varying them only slightly to meet the individual case. In the first place I always like to put them into the hospital for the period of adjustment to diet and insulin and to teach them certain fundamentals about their disease.

When the young diabetic gets into the hospital he is put at once on to a weighed diet containing, carbohydrate 200 grams, protein 1.5 to 2 grams per kilogram of body weight, and fat enough to make up the required calories. Growing children require from 50 to 75 calories per kilogram, depending on their state of nutrition when the diet is begun. Most of them, in the early weeks of treatment, need to be "fed up", but later on when normal weight has been resumed, their diets must be reduced to prevent overweight. Overweight must be assiduously avoided in diabetes, since it has been repeatedly shown to aggravate the disease.

My goal, then, in beginning adjustment of the young diabetic, is to give him an adequate diet immediately, high in carbohydrate and low in fat, according to Joslin's teaching that low fat increases the patient's ability to utilize sugar and helps to prevent some of the complications of diabetes.

All young diabetics need insulin. The dose is at first experimental, beginning with 3 to 5 units three times a day and increasing according to the urinary findings. And right here I would like to make a point which I believe most physicians, who have had no experience with diabetes, don't realize. It is possible, in fact better medicine, to adjust a diet to insulin without the use of frequent blood sugars. Blood sugars, while invaluable in the diagnosis of both hyperglycemia and hypoglycemia, are after all a fleeting picture of the amount of sugar in the blood at a given moment. One or two hours later, the situation may be entirely changed. Moreover, they involve the unpleasant procedure of sticking a needle into the child and for that reason should not, I believe, be done any more often than is necessary. And of course, they involve a specialized technique which is not available to many practitioners.

On the other hand, Joslin's four-period urine tests tell a much more striking story of the patient's ability to use the food given, with the help of the insulin, during that particular day. These specimens are passed before breakfast, about eleven in the morning, four in the afternoon and at bedtime. A simple test, the quantitative Benedict's reaction, is done on each one, the amount of urine measured, and thus an accurate determination of the sugar spilled is obtained. After observing these tests for a few days, it is a very simple matter to decide when to give the largest dose of insulin and how many doses are needed. I cannot overemphasize the value of this procedure in treating diabetic patients.

Diabetic diets are simple and easy to compute. I believe all children must have milk; about a pint and a half a day. They need raw as well as cooked vegetables and I think it wise to give them cod liver oil, to make sure they get the fat soluble vitamins in sufficient quantity. I never allow them to use saccharin for artificial sweetening because I believe it is much kinder to the child to get him over his "sweet tooth" as rapidly as possible, inasmuch as he is going to be deprived of sweets for an indefinite period of time.

I am going to give a sample of the diet of one of my diabetic children. I figure these diets using standard values from Joslin's diabetic card. I don't try to remember the amount of carbohydrate, protein and fat in the various foods. I remember only the general principles upon which I wish to base the diet, and then juggle the values until I get the desired result.

This boy, who is now on the diet, is fifteen years old, five feet five inches tall (illustrating the point which Priscilla White makes that diabetic children are tall for their ages) and weighs one hundred and three pounds. I want him to gain about fifteen pounds. Consequently I have him on a diet of carbohydrate 197 grams, protein 112 grams, fat 140 grams, calories 2700. He takes 35 units of insulin in the morning and 20 units at night. His diet is distributed as follows:

Breakfast		*Dinner and Supper*	
Shredded wheat	one	Potato	90 Gm.
White bread toasted	30 Gm.	Meat	75 Gm.
Egg	one	Vegetables (10%) (Raw twice weekly)	75 Gm.
Bacon	15 Gm.	Vegetables (5%) (Raw twice weekly)	150 Gm.
Cream	90 Gm.	Cream	30 Gm.
Grapefruit	200 Gm.	Butter	10 Gm.
Butter	10 Gm.	Orange	150 Gm.
		Cream cheese	15 Gm.

Lunch in *A.M.*, *P.M.* and *before bedtime*
One-half pint milk and two Uneeda biscuits

When I put this boy on his diet I made him stick to it just as it is. After I found out that he was conscientious and reliable, I allowed him to make several substitutions, to give him the idea that he could have variety and that I considered him responsible enough to have something to say about it himself. So I gave him the following list of equivalents:

One shredded wheat, equals—½ shredded wheat plus 50 Gm. banana.
One shredded wheat, equals—15 Gm. oatmeal (dry) plus 50 Gm. banana.
200 Gm. grapefruit, equals—100 Gm. orange, or 75 Gm. apple, or 75 Gm. blueberries, or 75 Gm. raspberries, or 75 Gm. of pears.
90 Gm. potato, equals—90 Gm. boiled rice, or 90 Gm. boiled macaroni, or 90 Gm. baked beans, or 90 Gm. green corn.

As soon as this boy gains the amount of weight he needs, I shall cut down on his fat until I get him on to a maintenance diet, which I expect will be about 100 grams of fat. His protein also is slightly higher than I usually give and that will be reduced when he has made the necessary gain.

One of the most important phases of the early hospital period is teaching the diabetic child to take care of himself. I feel that it is an obligation on the part of the physician to see to it that these boys and girls understand their disease. The older children, while in the hospital, can read Joslin's Manual for Diabetics and each day when the doctor makes a visit, one chapter or one phase of the subject can be discussed. It is amazing how interested they get and I have never seen a child become morbid as a result of the knowledge he acquires. He also has a chance to watch the preparation of his food in the diet kitchen and before he goes home he himself weighs it several times under supervision until he learns to do it accurately. He is taught how to measure his insulin, the most favorable spots for injections, and the care and sterilization of the syringe. I used to feel some trepidation when a ten year old first started to give himself insulin, but now that I know that children can do it with greater nonchalance and precision than adults, I have ceased to worry.

After the child has become accustomed to the everyday routine of his life as a diabetic, I teach him the two dangers which he is likely to encounter. These are coma and insulin reactions. I believe, with Priscilla White, that a diabetic should be "trained so thoroughly to avoid coma that, barring an infection, its development should be considered a disgrace". I try to make them understand, that after I have adjusted their diet and insulin, the responsibility is theirs and except for some unusual circumstance, my function from then on is advisory. They are their own doctors, I am merely the consultant.

I make a great point of intercurrent infections in my talks with these diabetic children, emphasizing the fact that any illness they may have will automatically make their diabetes worse while it lasts. For this reason they must avoid so far as possible exposure to contagious diseases of all kinds. And if they do at any time feel ill they must go to bed at once; take their insulin, unless the urine is sugar-free, even if they can't eat anything (this is very important because most people would omit insulin if they couldn't eat in the fear of insulin reaction); take an enema; drink copiously of hot liquids and send for me.

As a matter of fact I find that my young diabetics, who keep to their diets, are a most healthy group and seldom pick up the usual run of respiratory conditions which periodically go the rounds. I have never seen any explanation of this observation but probably it lies in the carefully supervised and controlled diet on which they live.

The other danger which the young diabetic is subject to is hypoglycemia. It is the rule rather than the exception and I tell them early that they undoubtedly will experience an insulin reaction sooner or later. They learn the symptoms which are nervousness, trembling, headache, double vision, sweating, and are told to drink the juice of an orange if any of these occur. They all carry a lump of sugar at all times and know that a reaction may come on suddenly at any minute, especially when they are taking excessive exercise.

When they have acquired the knowledge of their condition, they are ready to take their places in a normal world with very little handicap. The diabetic illustrates beautifully the truth of the saying "Knowledge is Power." I would not feel I had fulfilled my obligation to my patients, if I failed thus to arm them against the perils to which they are heirs.

Of course in the little children, one must educate the parents to take all the responsibility, teaching the child as fast as he is capable of learning. And that is much faster than anyone who has not had experience with young diabetics would believe. In Priscilla White's book on Diabetes in Childhood, there is a picture of a mere baby, two years and eleven months old, giving herself a dose of insulin!

In discussing the treatment of coma in the young diabetic there are certain things I have learned, more from experience than from books, which I would like to stress because I believe they will be helpful to the general practitioner. In the first place; since I have no laboratory technician available, and have to do my own blood sugars, I never stop to do one until I have the patient well started on his treatment. I much prefer to get him into the hospital, but failing that, I get a nurse to help me in the home.

Coma is an emergency demanding immediate and drastic treatment. The outcome is directly dependent on the rapidity with which treatment is instigated. The aims of treatment are directed toward overcoming the acidosis, combating the dehydration, and supporting the circulation. It is well to remember always that a patient does not die from sugar in the blood and urine. He dies, if he is allowed to, from the resulting acidosis, dehydration or circulatory failure.

Consequently, insulin is given to overcome the acidosis. By burning up the sugar circulating in the blood, it allows the incompletely oxidized acetone bodies to burn up also; a beautiful example of the graphic phrase; "Fat burns in the fire of the carbohydrates."

Insulin should be given, I believe, in comparatively small doses, but frequently. In a child who has been on insulin before, I give from ten to twenty-five units every half hour. In a child who has never taken insulin, I am more cautious, giving five units as the first dose and then ten or fifteen every half hour. The effect of these doses can be determined by the urine tests for sugar and acetone bodies (urines should be done every two hours during coma, catheterized if necessary) and by the character of the breathing. I have come to depend so much on the story the breathing tells me, when I am so busy treating coma, that I can't stop even to do urines, that I almost believe it possible to bring a patient successfully out of danger by observing that sign alone. The characteristic air hunger with its deep rapid breathing, which always accompanies diabetic acidosis, becomes gradually slower and less deep as the patient's condition improves. Insulin shock can never develop while air hunger is still present, nor can the patient become conscious again until it is improved. Thus it is possible to watch the effect of treatment and to increase or decrease the dose of insulin as needed.

Second only to insulin in importance is the giving of fluids to combat the dehydration which is always present. I believe it is never wise to depend on the oral route of administering fluids, since vomiting is easily induced and absorption uncertain. A hypodermoclysis of normal saline is begun immediately and from 500 to 1500 cc. are given, depending on the size of the child. Except in cases of collapse it is not necessary or wise to give intravenous fluids. If the child has not come out of coma and begun to take and retain fluid by mouth, the clysis should be repeated in six hours.

The next thing to be done is to wash out the stomach. I believe this procedure greatly adds to the patient's chances of recovery, for it prevents a frequent and distressing complication, that of acute dilatation of the stomach. Furthermore, it rids the stomach of all undigested food and thus shortens the vomiting period. I use normal saline, washing until the return is clear and repeating the procedure in four to six hours if vomiting, or dilatation, is still present.

A hot enema is always given early and repeated if necessary. These two measures, the lavage and the enema, help to restore the tone of the intestines which seem, sometimes, practically paralyzed. They should never be omitted.

While this drastic and hurried treatment is going on, the patient is handled very carefully and kept warm with blankets and hot water bottles. One should never forget that a person in diabetic coma is *in extremis*, fighting for life, and consequently must receive the gentlest possible treatment.

After the above measures have been properly dispatched, there is time to stop and take stock of the situation. The first question I ask myself is: How is the circulation standing the strain? A rapid, weak, thready pulse, possibly irregular, calls for stimulation. In that event, I give caffein sodium-benzoate and ephedrine sulphate by hypodermic every two hours alternately. I have learned from bitter experience that a patient may be successfully brought out of coma only to die from circulatory failure. My experience was with a young woman who had been in coma thirty hours before I saw her and, after eight hours of intensive treatment, recovered consciousness. However, her heart failed to respond to circulatory stimulation and she died twenty-four hours later from heart failure.

The second question I ask myself is: How are the kidneys standing the strain? As soon as I can, I do an albumin and a microscopic examination to gauge the amount of kidney damage or irritation. But more telltale in the emergency which exists, is the amount of urine put out by the patient. Every specimen passed or obtained by catheter, should be measured and tallied against the amount of fluid given by clysis. If, after six hours, the elimination by the kidney is defective, an attempt to produce diuresis can be made by giving a five per cent glucose solution subcutaneously or a twenty per cent solution intravenously. In the latter, the injection must be slow and the total fluid given, not more than 150 cc. in small children or 350 cc. in older children, because of the strain on the circulation.

Even if the kidney is sluggish in its output, one cannot stop giving fluids to a patient in diabetic coma, for an attempt must be made to overcome the dehydration, if life is to be saved. Sometimes, though not often, one seems to be between the Scylla of poor kidney function and the Charybdis of dehydration. There is no choice, however, but to go on giving fluids and hope that the kidney will respond.

As the child's condition improves and he begins to come out of coma, the interval between doses of insulin is lengthened, first to one hour, then to four. Careful watch must still be maintained, however, for lapses have often occurred. Frequent urinary sugar tests, with acetone and diacetic acid determinations, make it possible to judge the progress of recovery and to adjust the insulin accordingly.

At this stage I begin giving orange juice, ginger ale, clear broth, clear tea or coffee in small amounts (about an ounce at first) by mouth every hour. If retained, the amount is increased and the interval lengthened. Twenty-four hours after recovery from coma, I give a child half his usual diet but minus all the fat and watch his four period urine tests. The next day he gets his full diet except for the fat, and the third day he is restored to his pre-coma basis, but usually requires more insulin to keep sugar-free. The added insulin necessary I give in the middle of the day and try to drop it out as soon as possible.

With the employment of the above measures, I believe nearly all children in coma, or impending coma, can be saved. They are simple and can be used by anyone, even in remote districts where hospitalization is impossible. I would like to stress again the importance of the element of time in treating these cases. Every hour lost multiplies the chances of failure by a geometric ratio. And another point to be stressed is the absolute necessity of constant, watchful care on the part of the physician, until the fight has been won. I never leave a child in coma until he is safe on the road to recovery.

The question of surgery and diabetes demands careful attention. Whenever a surgical condition is superimposed on diabetes, the diabetes immediately becomes severe. A child with acute appendicitis, may go into coma in a few hours and the symptoms of the underlying cause of the coma be so masked, that a correct diagnosis would be impossible. To make the problem more difficult, the differential diagnosis between acute appendicitis and impending coma is often extremely obscure. Vomiting and abdominal pain are often present in both. A leukocytosis is present in both. To be sure, the abdominal pain and tenderness are more diffuse in coma than in appendicitis, but one cannot rely too much on this sign. I believe it wise to take into account a history of previous attacks, the fact that appendicitis associated with coma is rare compared with the existence of coma alone, and then, if a surgical abdomen is suspected, to make a small incision under novocain and take a look at the appendix. If pathology exists it can be remedied, if not, the patient suffers little from the procedure. This is the teaching of Joslin and his surgical associates, and is, it seems to me, as reliable as all the teachings from his vast experience are known to be.

In preparing the young diabetic for operation, it is always necessary to give extra insulin with glucose to insure a storage of glycogen in the liver, which can be called upon to combat the acidosis incident to the operation. If there is plenty of time to get the patient ready, I usually give him twice as much carbohydrate the day before his operation with half again as much insulin. I cut the fat out both before and after the operation. I do not try to keep him sugar-free, but concentrate on giving him plenty of available glucose to fight the acidosis which would certainly otherwise develop as the result of the anesthetic, the surgery, and the postoperative period of starvation.

After the operation, the diet is at first similar to that used for the child recovering from coma. Orange juice, oatmeal water gruel, ginger ale and skimmed milk, are allowed in small amounts, every fifteen minutes. The usual dose of insulin that the patient has been on, is divided into four hourly doses and given that way, irrespective of meals. Using the four period urine tests, one can judge the adequacy of this amount of insulin. If two successive tests are sugar-free, the next dose should be omitted and resumed only after two tests again show sugar.

In the days that follow, the diet is gradually increased, adding first the carbohydrate and protein constituents and leaving the fat to be added after the danger of acidosis is over. Diabetics, if thus protected, are good surgical risks and one should never hesitate to have necessary surgery done on a child because he has diabetes. In fact, I think the reverse is true—in the presence of diabetes, when surgery is indicated, the need is more urgent and acute than in a person whose carbohydrate metabolism is normal. This applies to the simplest conditions, such as infected tonsils and teeth, as well as to the more serious mastoids and appendices.

The young diabetic, subject though he is to infections, to surgery, to lapses of diet and to rapid and upsetting growth, can now be saved to live a normal and productive life. I know of nothing in all the practice of medicine so satisfactory to me as helping him to take his place in the world in spite of his handicap. Though I have had my troubles with the ones who insist upon skating along on thin ice for years, often getting me up at night to snatch them out of impending coma, never appearing to appreciate my concern for their lives, I still find that if I have the patience to put up with it long enough, they always come through in the end. Just the other day I had a letter from one of my young patients who is now away at school. He led me a merry chase for three years, during which time he was in and out of coma on the average of once in ten months. He never stuck to his

diet. He had a severe diabetes, requiring sixty-five units of insulin to cover his diet. Quite suddenly, when he was seventeen years old, after four years of diabetic irresponsibility, he turned over a new leaf and began to take care of himself. He stuck to it for a year and, when he wanted my sanction on going away to school, I gave it to him with justifiable confidence. His record at school couldn't be better, either diabetically or scholastically. Furthermore, he is a leader in the school, on the debating team, editor of the school paper, and in the orchestra. He doesn't think of himself as handicapped any longer and consequently has lost all the rebellion which used to make life so difficult for him. He does everything the other boys do, in fact "bummed" his way home for vacation to save the carfare to go to dances! The letter he wrote me was to tell me about winning a prize contest, sponsored by the Gorgas Institute in Washington. His ambition, at present, is to be a doctor.

I take no credit whatsoever for his change of heart. I am only thankful that I was "Johnny-on-the-spot" when he needed me in those trying adolescent years. The young diabetic deserves that from his doctor and I wish that more general practitioners would become interested in insuring the lives of these children with diabetes.

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MISCELLANY

RECENT DEATHS

PRATTE—Arthur A. Pratte, M.D., of Keene, died Monday, February 4, 1935, in Keene. He had practiced general medicine in Jewett, Conn., Marlboro, and Hinsdale in New Hampshire before going to Keene twenty-nine years ago, where he specialized in diseases of the eye, ear, nose and throat. Dr. Pratte was a member of the Board of Health for many years and health officer about a year. It was through his efforts that a full-time health officer was recently provided in Keene. He was a member of the Cheshire County Medical Society, and of the New Hampshire Medical Society of which he was a councilor.

TAYLOR—Fred B. Taylor, M.D., of Concord, died suddenly, January 24, at the Margaret Pillsbury Hospital. He was educated in the local schools, Andover Academy, Sheffield Law School of Yale University and Tufts College Medical School. He was affiliated with the New Hampshire Medical Society and was a member of the bar in both New Hampshire and Massachusetts. For five years he was physician at the State Prison and also served a term as a member of the Legislature.

COUNTY MEETINGS

The Belknap County Medical meeting was held at the Laconia Tavern, February 19. Dr. Francis Rackemann, Professor of Allergy at the Harvard Medical School, was the principal speaker. Dr. Rackemann talked on "The Cause and Treatment of Asthma and Eczema." His address was illustrated with lantern slides.

A special meeting of the Nashua Medical Society was held at St. Joseph's Hospital, February 19. The Society approved the stand of the American Medical Association in opposing the compulsory sickness insurance plans being studied by the President's Committee on Economic Security and went on record as opposing compulsory sickness insurance. Dr. Deering G. Smith, delegate from New Hampshire to the American Medical Association, gave a report of the meeting of the House of Delegates which was held February 15-16, in Chicago.

TUBERCULOSIS CLINICS

Dr. Robert B. Kerr of Manchester held a tuberculosis clinic at Laconia, February 11, and at Newport, February 15. One positive and two suspicious cases of tuberculosis were found among thirteen new patients examined at Newport. Improvement was noted in the general physical condition of many patients reëxamined.

CANCER CLINICS

During 1933 the State Cancer Commission allocated funds to the State Board of Health to establish free diagnostic cancer clinics. The Commission has designated the Elliott Hospital, Manchester; the Margaret Pillsbury General Hospital, Concord, and the Mary Hitchcock Memorial Hospital, Hanover, as treatment centers and the radium purchased by the Commission has been divided among these three hospitals, each having one or more physicians on the staff who have been trained and had experience in radium treatment.

NURSES

A report of visits made by nurses of the Keene District Nursing Association was submitted by the Superintendent of Nurses, Miss Anna C. Savage, at the quarterly meeting, January 9, 1935. Miss Savage reported 1950 visits during the past quarter. Of these, 539 were medical, 91 surgical, 149 obstetrical, 126 newly-born, and 145 welfare.

The annual meeting of the Meredith Public Health Association was held January 15 at the Nurses' Room. Miss Eva Heeney, District Nurse, made a report of

her work for the year, showing 861 regular visits and many others, advisory, prenatal, etc., making a total of 2807 visits.

Miss Della Robbins, Marlboro School and District Nurse, spoke on the health of the preschool child at a meeting of the Symonds preschool group, kindergarten and first grade pupils' mothers, January 25, at Keene.

Miss Velma V. Pettiner, Supervising District Nurse for the Portsmouth District Nursing Association, in her report for the month of January, announces the number of visits made as 222,—169 of these being on nursing work, 38 prenatal.

The February meeting of the Visiting Nurse Association of Rochester was held at the Board of Health room, February 22. Public health nurse report for the month of January showed 98 cases with 225 visits.

The report of the Nursing Service in Lincoln for 1934 made by Miss Addie Lessard, Public Health Nurse, was social service visits, 307,—total visits to homes, 1,729.

PERSONAL ITEMS

School teachers of West Swanzey, the Superintendent of Schools of the Swanzey district, State Commissioner of Education, James N. Pringle, and members of the Swanzey School Board joined in a dinner at Winthrop Lodge, Tuesday, January 29, to honor Dr. A. W. Hopkins of West Swanzey who has been a member of the town board of education for twenty-five years. The teachers presented Dr. Hopkins with a pen and pencil set and Mrs. Hopkins with a bouquet of flowers. Dr. Hopkins was primarily responsible for raising the Swanzey school system to its present high standing.

HOSPITALS

According to data compiled by the American Medical Association last year, New Hampshire has 45 registered hospitals with 4,735 beds and 325 bassinets, and 31,597 patients were admitted during the year. These figures do not include the thousands of out-patients who received treatment.

The widespread accomplishments and activities of the Hospital Aid Society of Cheshire County as an important aid to the Elliot Community Hospital and Keene Visiting or District Nurses' Association were reviewed at the annual meeting in the Nurses' Home recently. Mrs. Richard L. Holbrook was re-elected President. Miss Louise H. Thompson, Hospital Superintendent, expressed her personal thanks and that of the trustees and staff for the many extra services rendered by the society in the way of physical equipment, the furnishing of reading materials for patients and other helpful work.

"The chief accomplishment at the Laconia Hospital during the past year was the building of the east wing," states the annual report of Miss Lillian G. Williams, Superintendent, submitted at the annual meeting of the Laconia Hospital Association. The addition of the twenty-three bed wing increases the capacity of the hospital to eighty adult beds. During the past year, 2,179 patients have been treated.

NEW MEMBERS

J. Anderson, Somersworth.
Edna Walck, Dover.

DEFICIENT HOSPITAL AND HEALTH SERVICE

Thirty-one million persons in the United States live in areas which are seriously deficient in hospital and health services, according to a nation-wide study made by Alden B. Mills, managing editor of *The Modern Hospital*. The results of the study were published in the March issue of *The Modern Hospital*.

The Mills study continues one made last year by Michael M. Davis of the Rosenwald Fund, which revealed that about 1,300 of the 3,073 counties in the United States have no general hospitals at all.

The new survey points out that some counties do not need general hospitals since they are or can be served by hospitals in adjacent counties if the distance is not greater than fifty miles. Consequently in this study the United States is divided into areas with approximately fifty-mile radii.

After careful study of all factors involved it was decided that rural populations require a minimum of two hospital beds per thousand population although the study does not recommend or consider practical the building of general hospitals of less than twenty-five beds. For areas not requiring at least twenty-five-bed general hospitals, it is suggested that "cottage hospitals" or central medical service offices be established. This study also covers the distribution of physicians in the United States.

The study shows that there are 31,000,000 people and 29,000 physicians in areas that contain less than two hospital beds per thousand population and are more than fifty miles from a hospital center; there are 1,117,915 persons and 896 physicians in areas that have less than one-fourth of this ratio (0.5 hospital beds per thousand population); that there are 147 such 100-mile areas in the United States which have fewer than two hospital beds per thousand population; and that there is an actual need of 22,000 additional hospital beds in these 147 areas if minimum standards are to be met.

Individual states which have largest needs for additional beds are Texas, Alabama, Tennessee, Georgia, Mississippi, Kentucky, Missouri, Louisiana, Arkansas, Oklahoma, and North Carolina. Also there is revealed a serious deficiency of hospital facilities in some areas in western Kansas, parts of Virginia, South Carolina, Illinois, Ohio, Indiana, and Florida.

No large area deficient in hospital service exists in the New England States, according to this survey.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 21111

PRESENTATION OF CASE

A forty-eight year old American housewife entered complaining of epigastric discomfort.

She was first seen by her physician ten years before entry for symptoms that were associated with constipation and fatigue. Two years later she complained of epigastric fullness which often woke her at night, especially if she had eaten when fatigued. This sensation was relieved by induced vomiting or by Bell-ans pills. Complete x-ray studies were negative. When seen four years later, approximately four years before entry, she complained of indigestion and a dull pain beneath the sternum which were relieved sometimes by food, and always by soda or belching. She was frequently awakened at night by epigastric pressure and pain which were relieved only by vomiting. There was no hematemesis or food stasis. It was believed that her symptoms were associated apparently with undue tension and fatigue. Seven months before entry she reported again that she was well except for the epigastric discomfort which often woke her at 2 a.m. Constipation was quite marked and she had a good deal of gas daily. Vomiting still relieved the sensation of fullness. A simple régime helped her somewhat. A few weeks before entry she returned from Europe, having had a rough voyage associated with sea sickness. This was followed by an epigastric gnawing sensation which was relieved temporarily by food but which persisted, however, for several days.

Physical examination showed a well-developed and nourished, healthy-looking woman. Examination of the chest was negative. Abdominal examination was also negative. There was no tenderness or mass.

The temperature was 99.4°, the pulse 84. The respirations were 20.

Examination of the urine was negative except for an occasional white blood cell.

X-ray examination of the intestinal tract showed that the stomach filled in the usual manner. There was no change in peristalsis and no stasis. In the pyloric end of the stomach, close to the sphincter, there was a break in the outline with a small but distinct projection of barium in which there appeared to be the crater of an ulcerated area around which the barium had a mottled appearance. The sphincter was not deformed and the duodenal bulb appeared normal. Six hours after the meal the head of the barium had reached the splenic flexure.

On the fourth day an exploratory laparotomy was performed. Just above the pylorus on the posterior superior surface of the lesser curvature of the stomach there was a small amount of flexible thickening. A small crater may have been present, but it was very doubtful. The condition appeared so recent to the surgeon and there was so little evidence of an ulcer that it was considered unwise to do a resection at that time. It was felt that careful x-ray studies within the next three months was a more reasonable treatment than excision.

The patient's symptoms gradually disappeared under an ulcer régime and the stools were persistently negative for occult blood. Further x-rays three weeks later confirmed the previous observation. There was a definite variation from the normal on the lesser curvature close to the pyloric ring. Peristalsis was absent and a small crater could be demonstrated. A comparison with the previous films showed very little change. The crater-like deformity was possibly slightly smaller. Her symptoms continued to diminish. Another x-ray was done two weeks later. There was distinct change in the appearance of the prepyloric lesion. The crater had disappeared but the indurated area persisted. This could be demonstrated by failure of the peristaltic waves to pass over the lesser curvature side of the antrum and absence of the normal gastric rugae in this region. There was also a small residue at the end of six hours. Reëxamination with a spot film confirmed this finding and showed in addition a suggestion of an ulcer crater. Because of these findings a second operation was performed and the pylorus resected.

The patient was last seen ten months after operation and was in apparent good health.

CLINICAL DISCUSSION

DR. CHESTER M. JONES: She was an extremely neurotic individual, very unstable, fatigued very easily, and no doubt many of the symptoms of indigestion were functional in nature. However, indigestion occurring repeatedly at two o'clock in the morning must always be regarded from the point of view of organic disease. If the patient is awakened from a sound sleep by indigestion I think it has to be treated with greater respect than when noticed while awake.

On her arrival in this country she had a return of indigestion which had been absent while in Europe. This time she described it as "an epigastric gnawing sensation which was relieved temporarily by food but which returned, how-

ever, for several days." Her story was somewhat different at this time. It suggested an ulcer story more than at any other time in the history except for the statement a little previously that she had been awakened by pain in the early morning.

Physical examination did not help much. The numerous stool examinations were negative and the urine was also negative.

X-ray Interpretation

Dr. George W. Holmes: This is the x-ray film taken at the time of the examination, the report on which Dr. Jones has just read and here you can see the ulcer. You will notice that it projects beyond the outline of the stomach, has a characteristic crater, and a somewhat thickened margin, as shown by the decrease in the density of the barium at this point. The pyloric sphincter is apparently not involved and there is no change in the cap or the duodenal loop. The remaining portion of the stomach appears normal. I should like to call your attention particularly to the location of the ulcer. It is within one inch of the pyloric sphincter.

Clinical Discussion Continued

Dr. C. M. Jones: After the x-rays were taken the matter was considered by the doctor and he felt that it was advisable to consider surgery on the basis of an ulcerating lesion in the prepyloric region which might be malignant. That was agreed to and an operation was performed.

The patient had an uneventful convalescence after the operation and was placed on an ulcer régime at this time. The stools were negative. During this time it is important to note that the patient's symptoms had disappeared. She was free from symptoms at this time and subsequently, more so than she had been for many years.

Further X-ray Interpretation

Dr. Holmes: This film was taken as soon as the patient recovered from operation. You can see a slight deformity due to possible thickening around the margin of the ulcer, but the crater has disappeared. This film was taken a little later and shows practically no abnormality. There is possibly a little diminished density here. This is a film taken with the stomach filled. The next slide shows films taken with special technique to bring out the gastric mucosa. In this region you can see a definite abnormality of the mucosa in all the films taken.

Dr. C. M. Jones: Were those the last films taken, Dr. Holmes?

Dr. Holmes: Yes.

Further Clinical Discussion

Dr. C. M. Jones: The last films were taken two or three weeks after the second set of films and in spite of the fact that the patient was absolutely symptom free it seemed wise to reconsider the whole situation. At that time the subject was discussed again from the point of view that it was the prepyloric region with which we were dealing and that lesions shown by x-ray occasionally are difficult to feel at operation. We finally decided that in spite of the fact that there had been improvement in symptoms and in spite of the fact that there was improvement in the x-ray picture, there was still at the end of five or six weeks persisting evidence of a diseased condition in the prepyloric area. It was decided to operate again, this time with the decision to resect whether anything was palpable or not. I felt at the time that it was one of the very few occasions when a medical man has the right to ask a surgeon to perform a certain operation in spite of anything that may or may not be found at exploration. Certainly our experience in the past two or three years has finally accumulated to a point where we feel justified in saying that a lesion of the prepyloric region has to be operated on and resected. This patient was operated on a second time and resection was done.

Dr. Daniel Fiske Jones: They say that confession is good for the soul and I am glad to be here to make my own confession instead of having someone else do it. This is also an opportunity to apologize to the x-ray department for doubting their word. I should like to say also that in spite of the fact that I am confessing, I am going to confess that I will do it again when I find the same condition, and I am going to continue to doubt the x-ray department when I can demonstrate no pathologic lesion on surgical exploration; but I do apologize in this case.

This case was operated upon for the reason that Dr. C. M. Jones has just expressed, that one operates upon certain lesions of the stomach because of the location of those lesions. The doctor in charge and I decided that the patient ought to be operated upon. We had the x-rays and as you see on the plates the lesion was a very definite one and apparently was a deep ulcer. I am mentioning that because it has quite a bearing on what I am going to say. We operated upon this patient. I have felt a few stomachs and I think I should be able to feel certain things; but when you look at this x-ray picture and then go into the abdomen and find a stomach which is absolutely normal in appearance, which has a flexibility which is so slightly less than the normal stomach that you cannot say there is any lack of flexibility, when you cannot feel any ulcer at all, and when the only thing found is so slight that you say to your assistants, "There may be a bit of depression there," it is impossible for me to do a resection of such a stomach.

I appreciate that the location of a lesion in

this region is a serious thing, but I also appreciate that a subtotal resection of the stomach is a serious thing. It is impossible, I think, if I may answer Dr. Jones, for the medical man to say what should be done. He may say what ought to be done, but certainly the surgeon must say what he is going to do. I agree that all the information and all the help you can get from the medical man and roentgenologist are important and are worthwhile, but the surgeon is the man who is responsible and you cannot do resections of the stomach with no mortality. If you can, all right, go ahead and do as the medical man wants every time. I think he does not quite see the point of view of the surgeon. In some of these cases I think his opinion is of the greatest value. This patient had symptoms but when we compared my findings with the x-ray findings I was quite unwilling to take the picture which they showed me and pay no attention to what I found with my fingers and with my eyes. I therefore closed the abdomen deliberately and intentionally, telling the physician at the time that if our x-ray findings did not change or did not improve, or did not make us feel that everything was all right, then the patient could be operated upon again. If you operate and do a resection and you find that there is nothing and the patient dies, you cannot do anything. If, on the other hand, you close the abdomen and then find afterwards that it is necessary to operate, you can operate! That is one advantage. It is unpleasant for the patient I agree, but I think that you must take the reasonable point of view and not do an operation unless you can convince yourself that such a serious operation should be done, and I could not convince myself on the evidence found. I expect to do the same thing again some time if I live long enough and I shall do it without any hesitation, as much as I dislike to operate a second time upon a patient. There are many situations in surgery just like this and if you take the easy way and operate you will be a sad man, if you have any feeling at all, because you are sure to lose a patient some time when you should not have done anything. This patient was disappointed. She was not half so disappointed or half so uncomfortable as I was, but still I had not killed her and I left her in the hands of a man who would do a much better operation than I did. He did do it and she is alive and well for which I am thankful.

Dr. Leland S. McKittrick: Dr. Jones was not here when the problem came up again. The situation was accentuated by a matter that was not mentioned. Her mental state was such that a decision for or against operation had to be made at the particular time at which we did operate. She had become so upset by uncertainty that she had to know one way or the other.

I have learned a great deal from it and I have set down some suggestions which I would follow in another similar situation. It seems to me, in thinking it over, that probably one has to evaluate the risk of resection with the risk of delayed operation. Our experience has certainly been that prepyloric lesions are malignant until you prove them otherwise, and this experience would seem to show that in order to prove them otherwise at operation one must remove the pyloric end of the stomach. You cannot do that with all patients because of the risk of a partial gastrectomy. If in another case I were doubtful, I should begin medical treatment at once and follow the patient carefully. If there was not complete disappearance of the lesion in six weeks to two months, I should advise operation. If I operated, I should then feel obliged to treat it as cancer and do a resection.

Dr. George W. Holmes: First, I want to apologize to Dr. Jones. I should have been present at the operation. I think if I had been, I could have convinced him that there was something there, even if he could not feel it. I see his point very well. Here was a very definite lesion by x-ray which did not appear at operation. He had every reason to doubt the report. If I had been present at the operation I could have assured him that the report was correct.

Such a lesion as has been described we believe is a cancer until it is proved otherwise. This case is one of a considerable series of such cases, as has already been pointed out. This case also shows another important finding; namely, that an ulcerating lesion although cancerous may show marked improvement under dietary treatment or rest in bed. Certainly the ulcer was healed at the time the second operation was done. One of the signs that have been used by radiologists in differentiating benign and malignant gastric ulcers is the improvement of the patient under medical treatment, not only the symptomatic improvement but the appearance of the ulcer crater under medical treatment. If this case is properly interpreted that sign is of no further value.

Pathologic Diagnoses

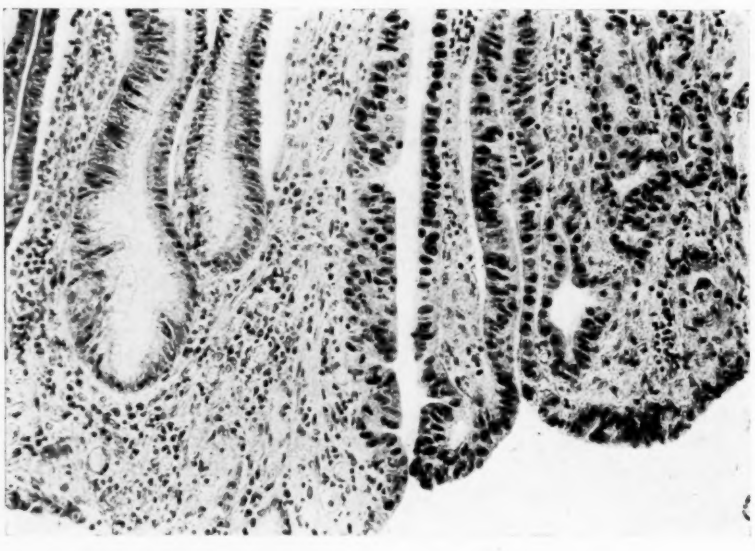
Carcinoma in situ.
Acute gastritis.

Pathologic Discussion

Dr. Tracy B. Mallory: I was present at the second operation and even after Dr. McKittrick delivered the prepyloric part of the stomach to me I was not able to feel anything abnormal. When we cut open the stomach it was evident that just behind the pyloric ring in the prepyloric region there was an area in the gastric mucosa that looked different from the rest. In this picture I think the artist has exaggerated the condition. The rugae came down and then

disappeared. There was not so sharp a difference in color between the areas. It was only after I looked two or three times that I really convinced myself that there was a lesion there. There were several minute erosions near the center which were covered with traces of fibrin, but nothing one could grossly call an ulcer. We felt that this was very much of a test case and therefore made particularly careful histologic studies. Dr. Bradley had serial sections of the entire area made. I will show you the pictures.

In cross sections of the area viewed under low power one can see normal glands on either side of the involved portion, in which the long parallel glands which would ordinarily run down to the muscularis mucosa have disappeared. At a slightly higher power one can localize the zone of abnormality a little more definitely. It is only the upper half of the mucosa that shows the change, whereas the deeper pyloric glands which resemble Brunner's glands are uninvolved.



A section through the margin of the atypical area in the mucosa.

Normally the cells covering the surface and lining the outer half of each gland are typical mucous secreting goblet cells. Under the conditions referred to as "gastritis" this mucous secretion ceases and the cells tend to become somewhat hyperchromatic, but they still show normal arrangement and the nuclei are always basally oriented.

In the picture which is now on the screen (see illustration) you see a sudden sharp transition from perfectly normal mucous secreting cells to highly atypical ones which tend to be piled three and four deep, to show no orientation of the nuclei, no trace of mucous secretion, and to be intensely hyperchromatic. The glands show a tendency to multiple lateral outpocketings and to papillary infolding, whereas the normal glands in this area have little tendency to branch till the deeper layers of the mucosa are reached. Mitotic figures—which are of course normal enough in moderate numbers in the gastric mucosa—are increased four or fivefold over the usual numbers. Throughout the entire area of involvement, a little over a centimeter and a half, we have not been able to find a trace of invasion anywhere. It would be difficult to call it established cancer; many histologists would not, since they insist upon invasion as evidence of established malignancy. On the other hand a degree of atypicality such as this is well recognized as related to malignancy. Some people will call these lesions precancerous, many other pathologists feel that they are already cancer. Broders, of the Mayo Clinic, has invented a name to cover the particular situation. He calls it "cancer in situ". I think that is a very appropriate term for what we have here. We have every characteristic of carcinoma except invasion. To show that this is not an isolated instance I will show you slides from another case. Although in this instance there is frank ulceration the mucosa on each side of the lesion shows the same type of abnormality as the preceding one. The findings can readily be duplicated from a series of gastric polyps some of which show the same suggestive evidence of beginning malignancy.

The overwhelming majority of these prepyloric lesions about which Dr. Holmes has spoken have been frank malignant cases. A couple have been obviously benign. These two are the only ones with any question of diagnosis. I have shown them to a great many pathologists and the average is three to one in favor of malignancy. A few stand out and say we cannot call it cancer until we get invasion. Whether at the moment we can call it cancer, I feel is academic. I do feel certain that it repre-

sents a stage on the way to cancer, the last stage before invasion and metastasis occur. Our only chance of curing cancer of the stomach is to operate on cases still in this stage.

CASE 21112

Presentation of Case

A forty-six year old American farmer entered complaining of epigastric discomfort.

At the age of seventeen, twenty-nine years before entry, he had an acute attack of burning pain across the upper abdomen. He was sick for about five days and spent the following month recuperating. No surgery was done. During the next ten years he felt fairly well except for an occasional sour burning in the pit of his stomach before noon. At the age of twenty-eight, eighteen years before entry, he was seen by a well known gastro-enterologist who found no organic disease. Two years later his appendix was removed. At operation his gall bladder was found to be grossly normal. About twelve years before entry his epigastric discomfort and burning increased in severity, often severe enough to wake him at night. Four years before entry he was seen by another well known gastro-enterologist who also found no evidence of organic disease. He was advised to reform his habits of irregular eating and sleeping. Two years before entry the epigastric burning became more severe and usually occurred between ten and eleven o'clock in the morning and eleven and twelve-thirty in the evening. These attacks were always relieved by soda or milk. He was seen again by the second physician. A gastro-intestinal series and a gastric analysis were negative. Approximately one year before admission, following three weeks of increased spasms of pain and rather strenuous exertion, he had a sudden attack of dizziness and weakness. He vomited occasionally but the vomitus never contained blood. The stools were black but did not contain any gross blood. He spent two weeks in a hospital where a gastro-intestinal series and barium enema were negative. The blood showed a red cell count of 2,800,000, with a hemoglobin of 55 per cent. Of three stool examinations one showed occult blood. When discharged his red blood cell count had risen to 4,000,000. During the past year his joint and epigastric cramps increased so that he was unable to work more than twelve days in two months. When he was seen three months before entry he had cramps all over his epigastrium, coming on in waves, lasting a few seconds and radiating to the chest. He also complained of easy fatigue for the past few weeks. Two months before entry he was put on a third stage gastric diet with tincture of belladonna which relieved about half of the symptoms. The cramps became less severe. He led a very easy life but had to take milk and toast or powders and milk almost every hour. He improved somewhat during the next two weeks, at the end of which period he could have his meals every two and a half hours rather than every hour. On the day before admission there was a slight increase in burning and spasm. A guaiac test on the stools was positive.

The family history is non-contributory.

He had been married twenty-two years, although he had not been living with his wife for some time. One daughter was living and well; another daughter, who was feeble-minded, died at the age of fourteen.

His past history is non-contributory except for severe intermittent migraine during the past twenty years.

Physical examination showed a well-developed and nourished man in no acute distress. The heart and lungs were negative. The blood pressure was 100/60. Abdominal examination was negative.

The temperature was 98°, the pulse 55. The respirations were 18.

Examination of the urine was negative. The blood showed a red cell count of 4,220,000, with a hemoglobin of 70 per cent. The white cell count was 5,500, 69 per cent polymorphonuclears. Only one out of seven stool examinations showed a positive guaiac test. The stools were soft and brownish-gray in color.

A gastro-intestinal series showed a 3.5 centimeter filling defect just below the midportion of the stomach. This filling defect was smooth except on the distal portion of the lesser curvature where it presented a scalloped margin. The defect appeared to lie on the anterior aspect of the stomach but satisfactory lateral views could not be obtained. Within the defect there was a one centimeter ulceration. No pedicle could be demonstrated and the surrounding mucosa was normal. The pyloric valve and the first portion of the duodenum were negative.

On the sixth day operation was performed. He had an uneventful convalescence and was discharged three weeks after operation.

Differential Diagnosis

Dr. George A. Leland: It would appear that this case is one of a man born in this country who at forty-six years of age had had indigestion for twenty-nine years. He had had repeated attacks of indigestion. These attacks had occurred specifically twenty-nine, eighteen, sixteen, twelve, four and two years ago and again several months, three months and two months before admission. These attacks seemed to point to some condition in the stomach or duodenum and were of the nature of, or suggested, some form of ulcer. His first attack lasted only five

days but required about a month for recovery.

He had some trouble on and off for the next ten years, and then eighteen years before admission, or eleven years after the onset, he was gone over very carefully by a gastro-enterologist who found no organic disease.

Two years later his appendix was removed and at that time his gall bladder was apparently observed by the surgeon and nothing made out grossly. That would be about sixteen years ago, before the time of the Graham test. The only laboratory test that might have been of avail at that time was the flat x-ray plate which, of course, was not very helpful. Nothing was mentioned about his stomach being palpated or inspected at that time. Perhaps that is just as well. The palpation of the stomach through an appendix-gall bladder incision is not necessarily productive of very startling results. Even the inspection of the stomach through a gall bladder incision is apt to lead one into error.

As these attacks went on they became not only more and more frequent in occurrence but also more and more severe. Epigastric burning relieved by soda or milk is distinctly an ulcer symptom. Two years before entry gastro-intestinal series and gastric analysis were performed and found to be negative. These points may be subject to some degree of debate. A well-taken gastro-intestinal series that was negative does not rule out a lesion in some obscure portion of the stomach. The negative gastric analysis makes one wonder whether that was an interpretation that the patient brought with him or whether it was checked up from its source. From looking that data over we have to take it for what it is worth and call it negative.

In the following year he had another symptom, bleeding. This was quite pronounced. The stools were black at that time. Another gastro-intestinal series was done and was negative. Although he never vomited blood, the black stools were undoubtedly due to blood, because his cell count became very low, less than 3,000,000, with a 55 per cent hemoglobin. Under care and rest in the hospital his blood came back to approximately normal.

"During the past year his joint and epigastric cramps increased so that he was unable to work more than twelve days in two months." We wonder if the "joint cramps" were thrown in to suggest hemorrhagic disease. There is nothing to suggest hemorrhagic disease in the subsequent history or examination. We do not know whether it was the joint cramps or the epigastric cramps that kept him from working. We are not informed but we will assume, since the rest of the history is gastro-intestinal, that it was the epigastric cramps.

"When he was seen three months before entry he had cramps all over his epigastrium, coming on in waves, lasting a few seconds and radiating to the chest." That would indicate hyperperistalsis. The radiation to the chest would seem to me to indicate that the peristaltic waves were high up in the gastro-intestinal tract rather than low down. As one might suppose with this chronic story of indigestion, hemorrhage and epigastric pain, he began to decline in health and to show fatigue, which is mentioned here. He was treated, and apparently quite adequately, two months before entry by a third stage diet and belladonna which relieved his symptoms, presumably, and diminished his hyperacidity, at the same time quieting down the hyperperistalsis; so, of course, the cramps became less frequent and less severe. He carried on quite well with toast, powders, etc. When he was admitted he was having another exacerbation of pain and again showing blood in the stools.

In the family history, the feeble-minded daughter I do not think brings up any particular question.

The past history is negative.

The physical examination is negative except for the low blood pressure. The abdomen was negative, ruling out any masses.

His blood picture was indicative of secondary anemia. There was one guaiac positive in several stool examinations.

Up to this point in a man of forty-six with several attacks of epigastric pain, showing evidence of hyperacidity, hyperperistalsis and hemorrhage, one would have to think very strongly of ulcer. At the age when his symptoms started one would think perhaps of some form of lymphoma; but we would not expect a lymphoma to last twenty-nine years without showing other manifestations. He might have started in with gastritis and worked up to a lymphoma, but then that is only in the realms of possibility rather than probability. Apparently he had something elusive because the repeated gastro-intestinal examinations by presumably competent roentgenologists were negative. That brings up the question of a lesion in some obscure part of the stomach or some obscure lesion like polyp that does not cause a great deal of distress but is not always so easy to find. Of course the question of syphilis comes up, but we do not know much about syphilis. There is nothing mentioned about the serological test. There are other obscure conditions of the stomach, neurofibroma, fibromyoma, and fibroma, but I do not think we can say there is anything characteristic in the story about them. In a man of forty-six who has had a long story, with a negative gastro-intestinal examination, we think of something in the stomach, and although the story suggests ulcer we have to keep in mind the possibility and probability of its having changed

into malignancy. So up to this point we would favor ulcer with the probability of its having changed to cancer in the more recent months or years.

Coming now to the gastro-intestinal series we find a three and a half centimeter, about an inch and a half, defect which is smooth except for a scalloped margin. Just what significance that latter term has in the roentgenological nomenclature I do not know. Perhaps Dr. Hampton will tell us. There was an ulcer there about a centimeter in diameter (that is about the size of a sixpence or a dime) and that size begins to come into the realm of malignant ulcers. The duodenum was negative. No mass was felt. We have no rectal corroboration of anything malignant. We are entirely adrift because of the absence of any mention of chemistry of the contents at this time. All we can say is that we have this scallop that may help us out. We have evidence of ulcer and if the scallop could tell us what kind of ulcer it is we might change our diagnosis from ulcer to cancer. In any event we would certainly advise operation. With a lesion on the anterior margin of the stomach we would expect to be able to remove a condition that had troubled him for twenty-nine years which may or may not have become malignant. Just what sort of repair we would make after this resection would depend entirely upon the condition found. I would undertake the operation with the expectancy of finding ulcer with beginning malignant changes.

X-ray Interpretation

Dr. A. O. Hampton: We were prepared for a very difficult examination when this patient came to the department because of his history and previous negative examinations. We were quite surprised to find a lesion. It was very obvious at the first glance that the stomach was abnormal, but the type of lesion was not nearly so obvious. The first swallow of barium was all that was necessary to do the entire examination. We learned no more after thirty minutes' effort than we knew at the first glance. There was a definite, round mass in the stomach with a definite localized area of ulceration in almost the exact center. The scalloped margin means a slightly wavy or nodular margin. I do not know what that means either; I just described it. I think a benign smooth tumor would not be scalloped. I do not know. Dr. Chester Jones was present and he asked me what I thought the lesion was and I said that my first impression was leiomyoma or leiomyosarcoma because the only other lesion I had seen similar to it was a leiomyoma. A large round tumor with a small central ulcer is very rare in my experience. After thinking about the scalloped margin, his history and the recent negative examinations, I decided that the tumor must have grown rapidly, especially since it had been missed before, because it was quite easy to see at our examination.

Clinical Discussion

Dr. Chester M. Jones: I have nothing to add except that his original symptoms on two occasions had been called nervous indigestion. He was an extremely highstrung individual who was most tense and just the type of person to have vague gastro-intestinal symptoms. It was obvious that he did have organic disease however, and it appeared that it was of more recent origin than was brought out by the history that Dr. Leland had. Before the x-rays were taken I was not at all sure what he had, but it seemed to me a little unusual for uncomplicated peptic ulcer, and because of the duration I had the same reaction, that malignancy should be considered. When I saw the x-rays with Dr. Hampton I felt sure in my own mind that it was leiomyoma; and it clinically fits in with the history of frequent repeated hemorrhages that one gets with leiomyoma. He had hemorrhages without any question. The thing that interested me was that this was a fairly good story for ulcer, not a perfect story, but a fairly good one in view of this lesion. I suppose it is due to the fact that he had an ulcerated lesion which produced symptoms in the same way as a benign peptic ulcer. I do not believe he had organic disease in the stomach for twelve or fifteen years. I believe it was of more recent origin, three or four years rather than more. The symptoms he had previously might well have been those of an unusually hard working, intense individual.

Dr. Arthur W. Allen: At operation this mass felt like a sessile polyp and it was easily palpated through the stomach wall. There were no glands that could be felt anywhere and no evidence of any involvement of the liver. The question of diagnosis had been thoroughly discussed before operation, agreeing with Dr. Leland that radical resection should be carried out regardless of the character of the tumor.

The history of the patient was very interesting to me. On the basis of its duration it was inconceivable that this lesion could have been going on for the long period of time that this man had had symptoms. I am not sure whether anyone knows when the beginning of a lesion of this sort would be and what symptoms might come from it. I do not believe we know whether he could have had a small ulcer there that healed and developed again repeatedly, so that perhaps this may have been the ultimate outcome of a chronic irritation.

The preliminary diagnosis which we made was leiomyosarcoma based on repeated hemorrhages and the x-ray appearance, because we had had one other similar case in which that diagnosis was proved pathologically. It looked

somewhat like this with the exception that the irregularity and the ulcerating area were not present.

To clear up one question which Dr. Leland raised as to the joint situation, I am sure we do not know what caused his shoulder pain. It was a transient affair, with negative x-ray findings, no limitation of motion and no localized tenderness in the region of the joint or bursa. At the same time he would have this pain in his left shoulder running down the arm, he had more or less pain in his chest and occasionally headaches. That he was a hypersensitive individual from a nervous standpoint there could be no doubt. The fact that he felt so well at the time of discharge from the hospital and insisted that he had not been so well for a great many years does not mean that all of his symptoms, gastric and otherwise, will remain relieved; but we hope he is rid of this particular lesion.

Clinical Diagnosis

Leiomyosarcoma of the stomach.

Dr. George A. Leland's Diagnosis

Ulcer of the stomach with probable early malignant change.

Pathologic Diagnosis

Spindle cell sarcoma of the stomach, probably neurogenic fibrosarcoma.

Pathologic Discussion

Dr. Tracy B. Mallory: The portion of stomach which was resected I have here and will pass around. There is a small localized tumor with a rather broad base projecting from the mucosal surface and showing a shallow ulcer near the center. The tumor extends through to the serosa and was readily felt from the outside. Its total diameter is under three centimeters. The gross appearance is typical of a myoma or fibroma of the stomach; that is, a tumor arising in the deeper layers of the stomach wall rather than in the mucosa. Most such tumors that have been reported (about 400 are on record) in the literature have been classified as myomas of varying degrees of malignancy. There have been, however, a limited number of fibromas, some of them very definitely of perineurial origin. Many of the authors content themselves with the diagnosis of spindle cell sarcoma, undoubtedly an indication that the distinction between myomatous and fibromatous tumors may be very difficult. When the tumor begins to grow rapidly and does not differentiate completely I think it becomes completely impossible in many instances. In this case the tumor is growing fairly rapidly, with numerous mitotic figures. It is not well differentiated, and we have had considerable difference of opinion in the laboratory as to how to classify it. Other pathologists have also disagreed. My personal impression is that it is a perineurial fibrosarcoma. I can show identical histologic pictures from acoustic neuromas for instance. The most striking feature of the tumor is rather long spindle cells with marked palisading of the nuclei, which is quite characteristic of the neurogenic type of fibrosarcoma, though it may occur in myomatous tumors.

The great majority of spindle cell tumors that have been reported have had a story similar to this. They have had vague ulcer symptoms, sometimes fairly typical, more often a little atypical. They have repeated gastric hemorrhages and usually it is a very long time before anyone succeeds in demonstrating them by x-ray. Any case of gastric hemorrhage in which a lesion cannot be demonstrated by x-ray is certainly open to suspicion as leiomyosarcoma of the stomach.

Dr. Allen: What are the probabilities of distant metastases?

Dr. Mallory: Undoubtedly the prognosis is distinctly better than that of carcinoma of that size. I do not think we can give a completely good prognosis however, though I should think his chances were better than fifty-fifty, which is far more than you can say for cancer.

Dr. Allen: In your opinion, is his past history in any way related to the tumor from the standpoint of actual pathology?

Dr. Mallory: I think it is almost impossible to say. The tumor seems to be growing fairly fast. I could not distinguish any core of older slowly growing tumor. On the whole the histologic appearances suggest that it is a rather fresh lesion. On the other hand the story of almost all these cases is very similar and runs back years and years.

Dr. Holmes: Do you think there was tumor there two years ago? Could you be reasonably certain of that?

Dr. Mallory: I do not think one could be certain. It is possible.

Dr. Benjamin Wood: I think the patient deserves a certain amount of credit because in spite of being told during the past five years that he had no organic disease he kept a very careful record of his symptoms. On his own initiative he had been doing guaiacs on the stools he passed for several weeks previous to the final hemorrhages and it was he who found the blood in his stools before he came in.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

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SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 Fenway.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The New England Journal of Medicine, 8 Fenway, Boston, Mass.

THE BOSTON HEALTH LEAGUE

IT is not uncommon to find that much of the most valuable work accomplished in any line of endeavor is done unostentatiously, with no faint suggestion of the trumpet's clarion call or the brave music of even a distant drum. The chief drawback to such inconspicuous service is that it has no sentimental appeal to open the purse strings of the community.

The very nature of the Boston Health League's organization, composed as it is of thirty-four agencies interested in health work in Boston, with representation from the Suffolk and Norfolk District Medical Societies and the Massachusetts Dental Society; and its activities, consisting of coördinating and advising in the work of these agencies, put it among those that work for the most part unseen.

A glance at the League's report for 1934, however, will reveal substantial evidence as to the value of its work and the scope of its activities.

The vexing question of domiciliary medical care for the City's indigent was definitely presented to the League for its advice, and it was the opinion of the Committee on Domiciliary Medical Care that the City should consider the question of assuming this responsibility. All parties concerned could not be immediately satisfied, but a temporary solution was effected.

The Executive Committee of the League in April took upon itself the responsibility of recommending to the Mayor the adoption of the recommendations of the Mayor's committee to survey the Boston Health Department. In substance the Committee recommended:

(1) That a permanent reorganization of the Health Department along modern lines as proposed by the Survey Committee should be undertaken.

(2) That much money could be saved by so doing without serious impairment of service.

(3) That any budgetary curtailment which may be decided upon for the Health Department should be based upon principles of effective public health administration.

(4) That a strong Advisory Council for the Health Department is essential.

The Educational Committee on Cancer gave nineteen lectures during the year and distributed literature; the Committee on Needs of Ward 9 held a Food Exhibit for two weeks in December in the Whittier Street Health Unit; at the request of the Executive Committee the Suffolk, Norfolk and Middlesex South District Medical Societies sent to their members reprints of the article published in The *New England Journal of Medicine* regarding the State Pneumonia Service.

The Committee on Social Hygiene sponsored a one day Institute for nurses on Syphilis and Gonorrhea, in coöperation with The Massachusetts Society of Social Hygiene and The Massachusetts League of Nursing Education. The Committee has continued sponsoring the Staff Council in Syphilis and Gonorrhea in coöperation with the Hospital Social Workers' Section of the Council of Social Agencies.

The Committee on Summer Camps again issued a pamphlet of suggestions for camp directors, and during the summer sent a score card to each camp requesting its return to the Health League Office in order that each organization might see how nearly it was complying with these standards.

A comparative study was undertaken on infant mortality in Charlestown and the West End in 1933, jointly by the Boston Council of Social Agencies, the Harvard School of Public Health, the Boston City Health Department and the Boston Health League to determine, if possible, causes for the high infant mor-

tality in Charlestown and the low mortality in the West End. The results pointed toward a need for the strengthening of medical care in Charlestown.

The Health League was asked to interpret the Health Survey of Boston which was included in the Unemployment Census of Massachusetts and indicated that there is more sickness and more use of free care in areas where unemployment is highest.

Rates for new cases and deaths from pulmonary tuberculosis, and infant mortality, have continued to be computed by Census Tracts and are on file in the office. The League has also furnished stenographic service and done other preliminary work necessary for the organization of the new Hospital Council.

There is nothing spectacular in these activities, certainly, but their value and the need for their continuance are apparent.

DR. WALTER R. BETT

Dr. Bett has recently been appointed librarian to the Medical School at Columbia University. Of British parentage, he received his medical training at St. Bartholomew's Hospital in London and at the London University Medical School. For many years he has been interested in medical history and was one of the instigators of the Osler Club of London, a group of young physicians who have kept the memory of Osler green. None of these young men ever knew Osler and yet they have succeeded in keeping alive a very active club, the principal event, each year, being the celebration of Osler's birthday by an appropriate address. The club, moreover, has collected many items about Osler, including a great many editions of his books and other material.

Among Dr. Bett's publications is an interesting paper on "Koplik's Spots", in the *British Journal of Children's Diseases* for April, 1931, one of a series of pediatric eponyms to be found in recent numbers of the same Journal. He also edited and contributed to "A Short History of Some Common Diseases", published in Oxford, 1934. Dr. Bett has served as secretary of the Section of the History of Medicine, Royal Society of Medicine, London, and, at the recent centenary meeting of the British Medical Association, he was secretary of the Historical Section. His brilliant mind and delightful sense of humor are well remembered by those who have attended the meetings of the Osler Club in London. It is a pleasure to welcome him to this country, for he will add much to the subject of medical history.

The Massachusetts Medical Society

ARRANGEMENTS FOR THE ANNUAL MEETING

The commercial exhibits at the Annual Meeting in June give every promise of providing an exceedingly interesting and instructive display. The Information and Registration Booths, and the Scientific and Commercial Exhibits together are to occupy the large Ballroom of the Hotel Statler. The concentration of these important units of the meeting in one room should work to the advantage and convenience of the attending physicians and the exhibitors.

The importance to the medical profession of the manufacturers and distributors of drugs, biological products and apparatus is recognized; and in these days of rapid scientific progress is increasing constantly. The accomplishments of many researches carried on in medical schools and hospital laboratories become available for practical application only through the facilities of commercial houses. Many real contributions have been made by workers in laboratories owned and operated by business organizations.

The Commercial Exhibition is not simply a group of booths hired by drug houses for the purpose of selling their products. It is a carefully selected presentation of ethical and acceptable materials for the scrutiny of the medical profession. Its immediate function is to *show* and not to *sell*. Your Committee of Arrangements would commend it to your attention in the hope that you will visit its displays and demonstrations which the coöperation of science and commerce has made available for everyday use in the practice of medicine.

SUB-COMMITTEES OF THE COMMITTEE ON PUBLIC RELATIONS

In order to bring attention to the important problems before the medical profession, the Committee on Public Relations of the Massachusetts Medical Society has formed several sub-committees which will proceed to study certain phases of economic and ethical questions. In order to secure facts on which to proceed, these several committees will need the coöperation of individual members of the Society who may have encountered perplexities which need adjustment.

Attention is called to a letter (The Problems of General Hospitalization) from the sub-committee to study the general hospitalization problems, which appears on page 493 of this issue. Many members of the Society have had some

experience in hospital management, or contact from the outside with these institutions, which may suggest adjustments from several angles. As indicated in the letter, there are matters which will appeal to many, and all who wish to participate in this study should place their ideas before this committee at an early date.

The report of the committee is awaited with interest.

THE MAIMONIDES MEETING

THE Boston Medical History Club will commemorate the eight hundredth anniversary of the birth of Moses Maimonides at the Boston Medical Library March 18, 1935 at 8:15 P.M.

The story of the life of this eminent Jewish physician and philosopher is of great interest and the address to be delivered by Professor H. A. Wolfson warrants a large attendance. The announcement appears on page 499 of this issue.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

CAREY, HELEN UPDEGRAFF. Ph.D. University of Michigan 1924. Associate in Biochemistry, University of Iowa 1928-1929. Special Research Chemist, New England Deaconess Hospital 1929-1932. Suddenly and unexpectedly died October 15, 1932.

HUNT, HAZEL M. A.B. Director of Chemical Laboratory, New England Deaconess Hospital, was associated with Mrs. Carey in the preparation of the article entitled "The Chemical Nature of Cataract in the Diabetic." Page 463. Address: 81 Bay State Road, Boston, Mass.

BENEDICT, EDWARD B. A.B., M.D. Harvard University Medical School 1923. Assistant in Surgery, Harvard Medical School and Massachusetts General Hospital. His subject is "Chronic Gastritis. A Clinical Discussion Based on Gastroscopic Examination." Page 468. Address: Massachusetts General Hospital, Boston, Mass.

BEATTIE, BARBARA. A.B., M.D. Cornell University Medical College 1924. Member of the New Hampshire State Board of Health. Her subject is "Some Experiences in the Treatment of Young Diabetics from the Point of View of the General Practitioner." Page 473. Address: Littleton, New Hampshire.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

THOMAS ALMY, M.D., *Chairman*, 140 Rock Street, Fall River, Mass.

C. J. KICKHAM, M.D., *Secretary*, 524 Commonwealth Avenue, Boston, Mass.

SHOULD A PATIENT WITH INTERMITTENT ATTACKS SYMPTOMATIC OF APPENDICITIS BE OPERATED UPON WHILE PREGNANT?

This question cannot be answered without some qualification.

It is generally agreed that in a genuine attack of acute appendicitis during any stage of pregnancy regardless of whether other attacks have preceded or this is the initial one, immediate appendectomy is the treatment of choice. A genuine attack of appendicitis must meet the diagnostic criteria of localized tenderness and abdominal pain over the region of the appendix, temperature, leucocytosis, nausea and vomiting, etc.

On the other hand, if the patient has a history of intermittent attacks simulating appendicitis or simply has a pain in the right side, which to the patient may be symptomatic of appendicitis, an operation under these circumstances during pregnancy cannot be justified. The ideal treatment, of course, in a patient with definite intermittent attacks symptomatic of appendicitis is removal of the appendix before pregnancy occurs.

If a patient with previous intermittent attacks develops an attack truly symptomatic of appendicitis during pregnancy, there is no way by which the physician can be assured that perforation and peritonitis may not take place. Wilson has shown that after perforation a mortality of fifty per cent occurs among pregnant women and that the danger to the mother advances in direct proportion to the stage of pregnancy. This is because the difficulty of diagnosis and of operative removal are increased in the latter months of pregnancy.

Obviously then, palliative treatment of an attack of appendicitis is even less indicated in the pregnant than in the non-pregnant woman.

In the attacks of appendicitis occurring early in pregnancy, appendectomy can be undertaken with little chance of interrupting the pregnancy if the uterus is carefully packed off, if no drainage is necessary, and if the patient is kept well morphinized during the early postoperative pe-

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

riod. Rupture of the appendix with the resulting local or general peritonitis usually results in abortion or premature labor.

In conclusion, the answer to the question is that a patient with intermittent attacks symptomatic of appendicitis should be operated upon immediately if she develops an acute attack during pregnancy.

MASSACHUSETTS LEGISLATIVE NOTE

H. 60. Bill (accompanying 59, recommendations of the Commissioner of Public Health) requiring the vaccination of children in private schools.

Report next annual session. Accepted in Senate. (Final.)

MISCELLANY

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1934 AND SEVEN YEAR AVERAGE

MONTH ENDING MARCH 2, 1935

	1935					1934			
Diseases	Week ending Feb. 9	Week ending Feb. 16	Week ending Feb. 23	Week ending March 2	Average cases reported for week corresponding to March 2 for past seven years	Week ending Feb. 10	Week ending Feb. 17	Week ending Feb. 24	Week ending March 3
Amebiasis	—	—	1	—	—	—	—	1	—
Cerebrospinal Men.	1	—	1	1	2	—	—	—	—
Chicken Pox	175	149	145	153	102	130	116	72	90
Conjunctivitis Inf.	1	—	—	—	—	—	—	—	—
Diphtheria	6	1	3	5	13	8	3	2	3
Dysentery Bacillary	2	1	—	—	—	—	—	—	—
German Measles	19	20	35	67	28	—	1	5	1
Influenza	9	21	12	32	753	18	3	6	24
Measles	617	620	689	785	247	33	39	30	49
Mumps	35	68	59	31	94	126	162	54	122
Paratyphoid Fever	—	1	—	—	—	—	—	—	—
Pneumonia (Broncho)	37	51	36	44	49	36	31	31	29
Pneumonia (Lobar)	33	52	37	38	56	63	37	46	39
Scarlet Fever	49	65	53	67	80	58	50	44	53
Septic Sore Throat	3	3	3	4	3	1	—	—	—
Smallpox	—	—	—	—	1	—	—	—	—
Trachoma	—	—	4	2	—	—	—	—	1
Trichinosis	—	—	—	—	—	—	—	—	1
Tuberculosis (Pul.)	34	21	14	30	27	20	24	11	41
Tuberculosis (O.F.)	1	1	2	2	3	—	—	4	4
Typhoid Fever	—	—	2	2	—	2	—	—	1
Undulant Fever	1	1	1	—	—	—	—	2	—
Whooping Cough	73	68	61	62	71	40	58	27	37
Gonorrhea	26	26	16	29	37	14	47	9	30
Syphilis	47	41	45	66	48	45	31	32	47

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

DR. STRONG ADDRESSES THE FLORIDA PUBLIC HEALTH ASSOCIATION

Dr. Richard P. Strong, Professor of Tropical Medicine of Harvard University, was the guest speaker at the recent meeting of the Florida Public Health Association in Jacksonville. The subject of his address was "Some Health Problems Regarding Parasitic Diseases Transmitted by Insects."

DR. PHANEUF GUEST SPEAKER IN PORTLAND, MAINE

Dr. Louis E. Phaneuf, Professor of Gynecology, Tufts College Medical School, addressed the Cumberland Medical Society, Portland, Maine, on the evening of February 28, 1935, his subject being "Cervical Cesarian Section—Indications, Technique and End-results."

AN ADDRESS BY DR. HORRAX

Dr. Gilbert Horrax, Director of the Division of Neurosurgery of the Lahey Clinic, Boston, Massachusetts, will talk on "Certain Neurosurgical Conditions Which Are Particularly Interesting to the Psychiatrist," on Tuesday, March 19, 1935, at 8:15 P.M., in the Reception Building of the State Hospital for Mental Diseases, Howard, R. I.

CORRESPONDENCE

THE PROBLEMS OF GENERAL HOSPITALIZATION

Editor, *New England Journal of Medicine,*

The undersigned have been appointed as a subcommittee of the Committee on Public Relations to study the problems of General Hospitalization, the Abuses and Competition of Hospital Out-Patient Departments, and the Hospitalization of the Workmen's Compensation Cases.

This sub-committee would appreciate having the members of the profession bring to our attention specific and typical instances of the abuse of the above types of hospital facilities. We particularly wish for information on the various ways of meeting the problem of hospitalization of the indigent outside of Metropolitan Boston with reference to the compensation received by the doctors. Constructive corrective ideas will be most welcome.

We suggest that your discussions be pertinent to the following problems:

1. Hospital out-patient departments.
2. Pay day or night clinics.
3. Consultation clinics for people of moderate means.
4. Varying degrees to which cities and towns pay the hospitals and doctors for the care of (a) indigent sickness; (b) indigent accident; (c) indigent contagious disease.
5. To what degree are the hospitals in your community following the recommendation of the Council of the Society that workmen's compensation cases be admitted only on a semi-private or private basis with compensation for the attending doctor?

Please address your letter to the chairman of the sub-committee at 45 Bay State Road, Boston.

J. HARPER BLAISDELL, *Chairman,*
FRANCIS H. DUNBAR,
CHANNING FROTHINGHAM,
WALTER A. LANE,
THOMAS H. MCCARTHY.

PYLEPHLEBITIS AND APPENDICITIS

Editor, *New England Journal of Medicine,*

The exceptionally fine and authoritative paper on "The Association of Pylephlebitis and Appendicitis" by Dr. Arthur W. Allen and collaborators, in your issue of January 31, 1935, proved of great interest to me. As in the case of Dr. Lund, who discussed the paper, it "brought up old times" and an anecdote that I can relate seems of some historical interest.

The elder Dr. Fitz published his rather epoch-making paper on the appendix, in 1896. Some time before that, I think probably about a year, Dr. Fitz made what I think was his sole visit to the City Hospital in the six years that I was there, on some errand of his own. I inveigled him into going on the ward and seeing a case that puzzled us. The patient was a girl of ten, entering at a late date in her illness, and we were not possessed of very much history. All we could see was enlargement of the liver, accompanied by some jaundice. We had not suspected the right iliac quadrant; nor, even after Dr. Fitz had made his diagnosis, did we find it more than unobtrusive.

Dr. Fitz made almost no examination but did suggest a pylephlebitis with a focal appendicitis, and such it proved to be. At autopsy a liver with multiple abscesses and an appendix with retropustulation. Dr. Fitz was then Professor of Pathology and undoubtedly surmised his diagnosis from pathological rather than clinical experience. This impresses the fact that to become a good clinician one must live both in the wards and the postmortem room. It is too often forgotten that clinical means the patient, alive and dead and that the actual seeing at the autopsy counts for vastly more than merely reading or hearing a protocol.

Thus this case was confirmatory of Dr. Allen's finding that this rare (1-300) combination seems confined to cases of appendicitis that have been either overlooked or received only late operation. In the discussion, Dr. Homans also related a case with autopsy disclosing an unrecognized appendicitis. In his case the "small abscess" was "retrocaecal". So in the one I have cited. Is there anything suggestive in this, apropos of Dr. Allen's finding of constant absence of thrombosis in the veins of the cecal region, thus, at the time of operation, at least, little basis for embolism or extension in the portal system?

Without remembering what was confirmatory, I have always carried the impression that, in my case, the extension of infection to the liver was posteriorly via the subserous connective tissue. In Dr. Allen's detailed case there was a large abscess in the left lobe and a "large" infrahepatic abscess, walled off, between the liver and the transverse colon. This it seems to me could scarcely be accounted for on the basis of a pylephlebitis. Certainly a large single abscess in the liver can be led up to through a connective tissue avenue. Thus in a very chronic duodenal ulcer observed, with a much thickened base, resting on the spine, there was a subphrenic abscess.

The connective tissue, significantly called areolar and cellular, cuts a large figure in the conduction of pus and of inflammatory infectious processes; as in the lung, rupture of an interlobular septum may, under certain conditions, produce a general subcu-

taneous emphysema. Thus in an old lady lying for a long period on her back with a wrong diagnosis of senile dementia, that same back was lined with pus from the pelvis to the neck.

In the cecal region before the days of operation and before Dr. Fitz organized our more or less vague conceptions, we were on the lookout for pus; but when it was pretty surely present, one couldn't get it operated on. Strange, because at that late date, conditions were simply those of a walled-off pus cavity requiring not an invasion of the abdominal cavity, but the incision of an abscess.

What antics pus in this region may cut up! A foul fecal discharge from the navel in a long-standing case! I admitted a right iliac case surgically for operation. I had traced pus over the rectum. Professor —— was the surgeon and the patient, with nothing observed, was about to be returned to the ward. I whispered to the interne "finger in the rectum". The bulging wall was found and then—trocar and cannula! The man recovered.

We had many right iliac inflammatory tumors in those days. I remember one as large as a cocoanut. I recall discussing the constituency of them with Dr. Fitz. A patient insisted upon going to work with one, the shape of a sausage, easily palpable. No harm came. Thus in those days we had practice in ferreting out some sequences in pathology not vouchsafed today. "Tempora mutantur et nos in illis" but, be it impressed, in the medical art there are no generations.

SAMUEL DELANO, M.D.

New Britain, Conn.,
February 13, 1935.

THE REPORTING OF SYPHILIS

The Commonwealth of Massachusetts
Department of Public Health
State House, Boston

March 6, 1935.

Editor, *New England Journal of Medicine,*

Since January 1, 1930, the Massachusetts Department of Public Health has distributed 244,095 grams of arsenicals (arsphenamine, neoarsphenamine and sulpharsphenamine) to physicians, clinics and institutions. During the last five years, 915 different physicians have received 43,534 grams, or 17.8 per cent of the total distributed.

Three hundred and forty-one (37.3 per cent) of these 915 physicians have never reported syphilis during these five years. It is evident, furthermore, from the amount of arsenicals ordered and the number of cases reported, that many of the 574 who have reported syphilis have reported only a few of their cases.

Two years ago the Department began enclosing its regular report forms with each package of arsenical sent to a physician, in the hope that reporting might thus be encouraged. Nothing whatsoever has come of this procedure, as will be evident from the following figures:

Year	Physicians Receiving Arsenicals	Have Never Reported Syphilis
1932	288	35.5%
1933	342	38.0%
1934	418	34.8%
1930-1934 (inclusive)	915	37.3%

Arsenicals are distributed for the treatment of syphilis. The law requires that syphilis shall be reported. The Department not only has made its report forms as simple as possible to fill out, but also provides envelopes for their return and pays the postage. It is regrettable that so many of those physicians who depend upon an expensive service rendered by the Department cannot, in turn, render the simple service, at no expense to themselves, of reporting syphilis. It is more to be regretted that their continued failure to report their cases must lead eventually to the ruling that no physician who has failed to report his cases will be supplied with arsenicals. Many states supply arsenicals only to those physicians who, with each request for the drug, indicate, in great detail, to whom the drug ordered is to be given.

Yours truly,
HENRY D. CHADWICK, M.D.,
Commissioner of Public Health.

OFFICIAL ACTIONS OF THE BOARD OF REGISTRATION IN MEDICINE

State House, Boston

THE SUSPENSION OF THE REGISTRATION OF DR. JULIUS LEVINE AND THE REVOCATION OF THAT OF DR. JOSEPH TESSIER

Editor, *New England Journal of Medicine,*

This is to inform you that at a meeting of the Board of Registration in Medicine, held February 28, 1935, it was voted to suspend for two months the registration of Dr. Julius Levine of 253 Norfolk Street, Dorchester, Massachusetts, for deceit in connection with automobile injury insurance claims.

At this same meeting, it was voted to revoke the registration of Dr. Joseph N. Tessier of 33 South Sixth Street, New Bedford, Massachusetts, for gross misconduct in the practice of his profession.

Yours very truly,
STEPHEN RUSHMORE, M.D., *Secretary.*

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
February 28, 1935.

Managing Editor, The *New England Journal of Medicine,*

In addition to the articles enumerated in our letter of February 4 the following have been accepted:

Merck & Co., Inc.

Tablets Cebione—Merck, 0.05 Gm.

Parke, Davis & Co.
Capsules Silvol, 6 grains
Kapseals Ortal Sodium with Amidopyrine

Pfanstiehl Chemical Co.
Aminoacetic Acid—Pfanstiehl

United States Standard Products Co.
Bermuda Grass Pollen Extract — U.S.S.P. Co.; Box Elder Pollen Extract — U.S.S.P. Co.; Burweed Pollen Extract—U.S.S.P. Co.; Careless Weed Pollen Extract — U.S.S.P. Co.; Cocklebur Pollen Extract — U.S.S.P. Co.; Corn Pollen Extract—U.S.S.P. Co.; Cosmos Pollen Extract — U.S.S.P. Co.; Cottonwood (Poplar) Pollen Extract — U.S.S.P. Co.; Dandelion Pollen Extract—U.S.S.P. Co.; Elm Pollen Extract — U.S.S.P. Co.; English Plantain Pollen Extract—U.S.S.P. Co.; Goldenrod Pollen Extract—U.S.S.P. Co.; Grasses Combined Pollen Extract — U.S.S.P. Co. (Bermuda Grass, June Grass, Orchard Grass, Red Top, Sweet Vernal Grass and Timothy in equal parts); Johnson Grass Pollen Extract—U.S.S.P. Co.; June Grass Pollen Extract—U.S.S.P. Co.; Lambs Quarters Pollen Extract — U.S.S.P. Co.; Maple Pollen Extract—U.S.S.P. Co.; Marsh Elder Pollen Extract—U.S.S.P. Co.; Mugwort (Wormwood) Pollen Extract—U.S.S.P. Co.; Orchard Grass Pollen Extract—U.S.S.P. Co.; Pigweed (Redroot) Pollen Extract—U.S.S.P. Co.; Ragweed (Common) Pollen Extract — U.S.S.P. Co.; Ragweed (False) Pollen Extract—U.S.S.P. Co.; Ragweed (Giant) Pollen Extract—U.S.S.P. Co.; Ragweed (Western) Pollen Extract—U.S.S.P. Co.; Ragweed Combined Pollen Extract—U.S.S.P. Co. (Giant and Common Ragweed, in equal parts); Red Oak Pollen Extract—U.S.S.P. Co.; Red Top Pollen Extract—U.S.S.P. Co.; Russian Thistle Pollen Extract — U.S.S.P. Co.; Rye Grass Pollen Extract—U.S.S.P. Co.; Sweet Vernal Grass Pollen Extract—U.S.S.P. Co.; Timothy Pollen Extract — U.S.S.P. Co.; White Ash Pollen Extract — U.S.S.P. Co.; White Oak Pollen Extract—U.S.S.P. Co.

The following product has been accepted for inclusion in the List of Articles and Brands Accepted by the Council But Not Described in N.N.R. (New and Nonofficial Remedies, 1934, p. 443):

Sharp & Dohme
Ampules Sodium Cacodylate — Mulford, 7½ grains, 5 cc.

Yours sincerely,
PAUL NICHOLAS LEECH, *Secretary*,
Council on Pharmacy and Chemistry.

THE HINTON TEST IN NEUROSYPHILIS*

January 12, 1935.

Editor, *New England Journal of Medicine*,

In a letter[1] published in The *New England Journal of Medicine*, March 29, 1934, it was announced by the State Department of Public Health that the Wassermann Laboratory would henceforth perform the Hinton test instead of the Wassermann test on all specimens of blood submitted for the serological detection of syphilis. This decision was reached after comparative studies of various serological tests were made at the Wassermann Laboratory and the Boston Dispensary and was the culmination of Dr. Hinton's serological investigations covering a period of several years[2], [3], [4].

The Hinton reaction is based on the principle of precipitation in contradistinction to the Wassermann complement fixation methods. As a result of a comparison of the tests performed on thousands of bloods, it became apparent that the Hinton test was an exceedingly sensitive one and it was hailed as a distinct advance in the serodiagnosis of syphilis. The serological data on the blood sera of patients in the Boston Psychopathic Hospital obtained routinely during the past few years present sufficient evidence of the high degree of sensitivity of the Hinton test in the detection of syphilis.

In the course of Dr. Hinton's investigations, interest became centered upon the serological reactions in cases of neurosyphilis. Many observations seemed to indicate that a negative Hinton reaction on the blood ruled out the presence of neurosyphilis, and conversely that neurosyphilitic cases invariably showed a positive blood Hinton reaction. This soon led to the conclusion that lumbar punctures could be eliminated and Hinton tests substituted in the serodiagnosis of neurosyphilitic patients. It is the purpose of this communication to discuss this aspect of the serological problem and to present briefly some observations recently made on patients in the Neurosyphilis Clinic, which serve to illustrate some of the vagaries of the Hinton test in neurosyphilis. These observations were derived from the serological data obtained during the past year and a half. From the standpoint of technique, therefore, the results represent the most recent refinements of the Hinton test and consequently would yield reliable data upon which conclusions may justifiably be drawn.

For many years it has been generally conceded that the status of the spinal fluid is the most accurate index of activity of the neurosyphilitic process. Moreover, lumbar puncture has become an established procedure in the treatment of neurosyphilis and the results of spinal fluid examinations furnish the best criteria for the continuation or cessation of treatment. These tenets have been stressed repeatedly by Dr. H. C. Solomon, who has, in the course of twenty years, accumulated considerable data on the value of lumbar puncture in neurosyphilis. This evidence indicates that the best therapeutic results are obtained by periodic check-ups of the spinal fluid and that the underlying disease may be said to be arrested when a completely negative spinal fluid has been maintained. These are funda-

*From the Boston Psychopathic Hospital.

mental principles in the treatment of neurosyphilis and must be adhered to in the application of the Hinton test.

There are two questions, then, that must be answered: 1. Do cases of neurosyphilis always show a positive Hinton reaction on the blood? 2. Does the Hinton test become negative when the spinal fluid tests return to normal during the course of treatment? In order to throw some light on these questions, Berk and Hinton[5] made a comparative study of the spinal fluid examinations and the blood Hinton reactions in 787 cases in which there had been infection by syphilis, and concluded that a negative Hinton reaction on the blood "almost totally excludes the likelihood of there being laboratory evidence of neurosyphilis obtainable by lumbar puncture". It is to be noted that the Hinton test was modified so that it would not give a negative result when the spinal fluid was abnormal.

From the very beginning of Hinton's investigations on the precipitation reactions of blood sera, comparative serological data have been accumulated on all cases in the Psychopathic Hospital Clinic, and sufficient experience with the results of the Hinton test has been obtained. However our observations have not justified the elimination of lumbar puncture in the management of neurosyphilis, and the usual custom of making routine spinal fluid examinations has continued. A brief summary of illustrative findings follows:

1. In a group of untreated cases of neurosyphilis with strongly positive spinal fluids there were found eleven patients with frank clinical signs of paresis who showed consistently negative Hinton reactions on the blood. This number is not large, but it indicates definitely that lumbar puncture is indispensable in the serologic diagnosis of neurosyphilis and that a negative Hinton test does not rule out the presence of syphilis of the nervous system. The total number in this series is drawn from the routine admissions to the hospital during the one and one-half year period and may be estimated at three hundred neurosyphilitic cases.

2. Among the treated cases there were seven patients who after considerable treatment showed negative Hinton reactions on the blood but still maintained positive spinal fluids of varying degrees of strength. This would indicate that even from the treatment standpoint the Hinton test is not a reliable criterion for the continuation or cessation of therapy in cases of neurosyphilis.

3. Conversely in the group of treated cases there were five cases in which the Hinton tests on the blood were positive. These patients were previously treated and maintained negative spinal fluids over a period of years during which no treatment was given despite the finding of positive Hinton reactions on the blood. It is possible that these cases represent instances of active syphilis. But from the neurosyphilitic standpoint the persistence of a positive Hinton test is of no greater significance than other positive blood tests, such as the Wassermann, which sometimes persist long after the spinal fluid is made negative by treatment. Whether or not these patients should be treated is a difficult question to decide. But it is to be pointed out in a considerable experience with the serological reactions in cases of neurosyphilis that the blood reactions are of no significance in the prognosis and final result of treatment.

4. In the course of intensive treatment of neurosyphilitic cases carried out over a period of years, there were found thirty-eight cases in which a negative spinal fluid was obtained and which still showed positive Hinton reactions on the blood. All of these patients were arrested paretics with varying degrees of mental defect and were still being treated because of the general policy of maintaining the negative result. There are no indications of reversal of the blood Hinton reactions to normal, although there is sufficient evidence in these cases of arrest of the neurosyphilitic process.

Finally, mention should be made of the patient who refuses lumbar puncture because he has been told that the Hinton test on his blood is negative and, therefore, believes that his spinal fluid must be negative also. It is not an uncommon experience in our clinic to encounter such patients with frank clinical signs and symptoms of neurosyphilis.

Very truly yours,

SAMUEL H. EPSTEIN, M.D.

74 Fenwood Road,
Boston, Mass.

REFERENCES

1. New Eng. J. Med. 210:711, 1934.
2. Hinton, W. A.: Boston M. & S. J. 196:993, 1927.
3. Hinton, W. A., and Berk, A.: J. Ment. Sc. 77:495, 1931.
4. Hinton, W. A.: J. Lab. & Clin. Med. 18:198, 1932-33.
5. Berk, A., and Hinton, W. A.: Am. J. Syph. & Neurol. 18:92, 1934.

RECENT DEATHS

MARTIN — FRANKLIN H. MARTIN, M.D., Director General of the American College of Surgeons, died in Phoenix, Arizona, March 7, 1935. Dr. Martin was seventy-seven years old. He suffered an attack of coronary thrombosis, and although he was reported in the newspaper to have been in previous good health, his friends had felt concern over his physical condition when he was in Boston at the time of the last meeting of the American College of Surgeons.

Dr. Martin was essentially an organizer, having founded the clinical Post-Graduate Medical School in 1882 with Dr. G. W. F. Coleman, and the *American Journal of Surgery, Gynecology and Obstetrics*. This publication was donated to the American Surgical Association in 1932 together with the building and its contents located in Chicago.

Dr. Martin was active during the World War on the Advisory Commission of the Council of National Defense.

DUANE—WILLIAM DUANE, M.D., formerly Professor-Emeritus of Bio-Physics at Harvard, died in Philadelphia, March 7, 1935.

He was associated with Boston physicians in x-ray research for several years, and was consulted when technical matters required elucidation. When the Abrams propaganda was under way in Boston, his advice enabled doctors to refute the exaggerated claims of this cult.

LAWRENCE—ROBERT MEANS LAWRENCE, M.D., of 117 Bay State Road, Boston, died at his home, March 7, 1935. He was born in Boston in 1847, the son of William R., and Susan (Dana) Lawrence, and the grandson of Amos Lawrence, the philanthropist.

After graduating from Harvard College, he entered the Harvard Medical School and graduated in 1873. He joined the Massachusetts Medical Society in 1873, and resigned in 1912.

He was an athlete in early life, with especial interest in football.

He was connected with the Boston Dispensary for ten years, later serving as trustee, and was surgeon of the first regiment of Massachusetts Volunteers. His religious affiliations were with the Episcopal church.

Two daughters, Mrs. Isabel B. deGersdorff, of Bedford Hills, New York, and Miss Madeleine Lawrence, of Boston, survive him.

The Massachusetts Medical Society

SECOND ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The spring sessions of the 1934-'35 schedule will be given in the various districts on the dates listed below:

Barnstable

March 17 — Obstetrics and Gynecology (First Session).
March 24 — Obstetrics and Gynecology (Second Session).
March 31 — Obstetrics and Gynecology (Third Session).

Berkshire District

April 4—Obstetrics and Gynecology (Third Session—Gynecology).
April 11—Endocrinology (Second Session).
April 18—Endocrinology (Third Session).
April 25 — The Common Neuroses and Their Treatment in Private Practice. The Psychoses—Early Diagnosis.
May 2—Dermatology and Syphilis.

Bristol North (Attleboro Section)

March 19—Endocrinology (First Session).
March 26—Endocrinology (Second Session).
April 2—Endocrinology (Third Session).
April 9—Cardiovascular Disease (First Session).
April 16 — Cardiovascular Disease (Second Session).
April 23 — Cardiovascular Disease (Third Session).

Bristol North District (Taunton Section)

March 20 — Cardiovascular Disease (Third Session).
March 27—Dermatology and Syphilis.
April 3 — Obstetrics and Gynecology (First Session).
April 10 — Obstetrics and Gynecology (Second Session).
April 17—Obstetrics and Gynecology (Third Session).

Bristol South (Fall River Section)

March 18—Surgery (First Session).
March 25—Surgery (Second Session).
April 1—Surgery (Third Session).

Bristol South District (New Bedford Section)

March 22—Endocrinology (First Session).
March 29—Endocrinology (Second Session).
April 5—Endocrinology (Third Session).
April 12—Cardiovascular Disease (First Session).
April 19—Holiday—no session.
April 26—Cardiovascular Disease (Second Session).
May 3—Cardiovascular Disease (Third Session).
May 10—Obstetrics and Gynecology (First Session).
May 17 — Obstetrics and Gynecology (Second Session).
May 24—Obstetrics and Gynecology (Third Session).
May 31—The Common Neuroses, etc.

Essex North District

April 2—Obstetrics and Gynecology (First Session).
April 9—Obstetrics and Gynecology (Second Session).
April 16—Obstetrics and Gynecology (Third Session).

Essex South District

March 19—Dermatology and Syphilis.
March 26 — Cardiovascular Disease (First Session).
April 2 — Cardiovascular Disease (Second Session).
April 9 — Cardiovascular Disease (Third Session).

Franklin District

March 27—Dermatology and Syphilis.
April 10—Cardiovascular Disease (First Session).
April 17—Cardiovascular Disease (Second Session).
April 24 — Cardiovascular Disease (Third Session).
May 1—Obstetrics and Gynecology (Third Session).

Hampden District (Springfield and Holyoke Sections)

March 21—Endocrinology (Second Session).
March 28—Endocrinology (Third Session).
April 4—The Common Neuroses, etc.
April 11—Endocrinology (First Session).

Hampshire District

April 17—Surgery (First Session).
April 24—Surgery (Second Session).
May 1—Surgery (Third Session).
May 8—Cardiovascular Disease (First Session).
May 15 — Cardiovascular Disease (Second Session).

May 22 — Cardiovascular Disease (Third Session).

Middlesex East District

March 20 — Cardiovascular Disease (First Session).
March 27—Cardiovascular Disease (Second Session).
April 3—Cardiovascular Disease (Third Session).
April 10—Surgery (Third Session).
April 17—Dermatology and Syphilis.

Middlesex North District

March 29 — Cardiovascular Disease (First Session).
April 5 — Cardiovascular Disease (Second Session).
April 12 — Cardiovascular Disease (Third Session).
April 19—Holiday—no session.
April 26—Amebiasis and Parasite Diseases Common in New England.

Norfolk District (Faulkner Hospital Section)

March 25—Surgery (Third Session).
April 1—Obstetrics and Gynecology (First Session).
April 8—Obstetrics and Gynecology (Second Session).
April 15—Obstetrics and Gynecology (Third Session).
April 22—The Common Neuroses, etc.

Norfolk District (Norwood Section)

April 26—The Common Neuroses, etc.
May 3—Obstetrics and Gynecology (Third Session).
May 10 — Cardiovascular Disease (First Session).
May 17—Cardiovascular Disease (Second Session).
May 24—Cardiovascular Disease (Third Session).

Worcester District (Milford Section)

March 28—Dermatology and Syphilis.
April 4—Surgery (First Session).
April 11—Surgery (Second Session).
April 18—Surgery (Third Session).
April 25—Obstetrics and Gynecology (First Session).
May 2—Obstetrics and Gynecology (Second Session).
May 9—Obstetrics and Gynecology (Third Session).
May 16—Cardiovascular Disease (First Session).
May 23 — Cardiovascular Disease (Second Session).
May 30—Cardiovascular Disease (Third Session).

Worcester District (Worcester Section)

April 3—Dermatology and Syphilis.
April 17—Endocrinology (Third Session).
April 24 — Cardiovascular Disease (First Session).
May 1 — Cardiovascular Disease (Second Session).
May 15 — Cardiovascular Disease (Third Session).

Worcester North District (Ayer Section)

March 21—Surgery (First Session).
March 28—Surgery (Second Session).
April 4—Surgery (Third Session).
April 11—The Common Neuroses, etc.

Worcester North District (Fitchburg Section)

April 12 — Cardiovascular Disease (First Session).
April 19—Holiday—no session.
April 26—Cardiovascular Disease (Second Session).
May 3—Cardiovascular Disease (Third Session).
May 10—Dermatology and Syphilis.

NOTICES OF MEETINGS

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, March 21, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

The next meeting of the New England Ophthalmological Society will be held on Tuesday, March 19, 1935, at the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston.

PROGRAM

9:00 A.M. Clinic and operating room.
11:30 A.M. Neuro-ophthalmological conference.
4:00 P.M. Clinico-pathological conference.

Evening Program, 8:00 o'clock:

Paper: Does Diabetes Contribute to Cataract Formation?

1. Morphological evidence. Dr. J. H. Waite.
2. Chemical evidence. Miss Hazel Hunt.

Discussion. Dr. Y. Subba Row.

DR. BENJAMIN SACHS, *Secretary.*

LAWRENCE CANCER CLINIC

Established 1928

Lawrence, Mass., March 6, 1935.

To the Physicians of the North Half of Essex County:

Dear Doctor:

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, 1 Garden Street, Lawrence, upon Tuesday, March 19, at 10:00 A.M., will

be a Demonstration Clinic with Channing C. Simmons, M.D., of Boston, Associate in Surgery at Harvard University Medical School, Surgeon-in-Chief to Collis P. Huntington Memorial Hospital, and member of the Cancer Commission of Harvard University, Boston, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This service is gratis. Your attendance at the Clinic is always welcome.

This Clinic is endorsed by the Committee on Postgraduate Instruction of the Massachusetts Medical Society.

Committee
ROY V. BAKETEL, M.D.,
CHAS. J. BURGESS, M.D.,
FRED'K D. MCALLISTER, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D., *Chairman.*

BOSTON MEDICAL HISTORY CLUB, BOSTON MEDICAL LIBRARY, GREATER BOSTON MEDICAL ASSOCIATION

BOSTON MEDICAL LIBRARY, 8 FENWAY, MONDAY, MARCH 18, 1935, AT 8:15 P.M.

MAIMONIDES MEETING

In commemoration of the 800th anniversary of the birth of Moses Maimonides (1135-1204), greatest of Jewish physicians and philosophers.

An address will be given by Professor Harry A. Wolfson, who is Nathan Littauer Professor of Jewish Literature and Philosophy at Harvard University.

Title: "The Relation Between Medicine and Philosophy in Maimonides."

You are cordially invited to attend the meeting.

JAMES F. BALLARD, *Secretary,*
Boston Medical History Club.

BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, March 15, 12-1, Cheever Amphitheatre.

Dr. Elliott C. Cutler, Professor of Surgery at the Harvard Medical School, and Surgeon-in-Chief, Peter Bent Brigham Hospital, will discuss "The Significance of Vomiting."

Physicians and medical students are invited.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, March 26, at 8:15 P.M.

PROGRAM

Presentation of Cases.

The Circulation in Normal and Hypertrophied Hearts. By Dr. Joseph T. Wearn, Professor of Medicine, Western Reserve Medical School, Cleveland, Ohio.

The Establishment of Collateral Blood Channels to the Heart by Operation. By Dr. Claude S. Beck, Associate Professor of Surgery, Western Reserve Medical School, Cleveland, Ohio.

MARSHALL N. FULTON, M.D., *Secretary.*

ACADEMY OF PHYSICAL MEDICINE

Annual Meeting, The Claridge Hotel, Atlantic City, New Jersey, June 12 and 13, 1935. For further details address: Arthur H. Ring, M.D., Secretary-Treasurer, Arlington, Mass.

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held in the Evans Building at the Massachusetts Memorial Hospitals, 82 East Concord Street, Boston, Monday, March 25, at 8:15 P.M.

PROGRAM

(1) Some Observations Upon Palpitation. W. D. Reid, M.D.
(2) Primary Bilateral Pulmonary Thrombosis. M. A. Lesser, M.D.

Discussion by S. McGinn, M.D., and C. F. Branch, M.D.

(3) A Transitional Region Between Skeletal and Cardiac Muscle. F. H. Pratt, M.D.
(4) An Oscillograph for Electrocardiography. W. D. Reid, M.D.
(5) Roentgenographic Study of the Heart. A Moving Picture of the Heart. G. Levene, M.D.

JAMES M. FAULKNER, M.D., *Secretary.*

ROBERT BRECK BRIGHAM HOSPITAL

CLINICAL MEETING

An open Clinical Evening will be held at the Robert B. Brigham Hospital, 125 Parker Hill Avenue, Boston, on Tuesday, March 19, at 8:15 P.M.

There will be a presentation of cases and open discussion on "The Diagnosis and Treatment of Various Types of Chronic Arthritis."

Physicians, nurses, and students are invited to attend.

The usual Wednesday, 4:30 P.M., meeting will be omitted for that week.

TUFTS MEDICAL ALUMNI DINNER

The annual dinner of the Tufts College Medical School Alumni Association will be held Wednesday, March 20, 1935, at 6:30 P.M. in the main dining room of the University Club, Boston. The annual business meeting will take place at the University Club at 5:30 P.M. This year special prominence will be given to the Classes of 1895, 1900, 1905, 1910, 1915, 1920, 1925, and 1930. The speaking will consist of short talks by representatives of these classes, Dr. Alonzo K. Paine presiding.

AMERICAN HEART ASSOCIATION, INC.

The Eleventh Scientific Session of the American Heart Association will be held on Tuesday, June 11, 1935, from 9:30 to 5:30 P.M. at the Hotel Claridge, Atlantic City, N. J. The program will be devoted to various subjects on cardiovascular disease.

GERTRUDE P. WOOD, *Office Secretary.*
50 West 50th Street, New York, N. Y.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MARCH 18, 1935

Monday, March 18—

*8:15 P.M. Boston Medical History Club, Boston Medical Library, and the Greater Boston Medical Association. Boston Medical Library, 8 Fenway.

Tuesday, March 19—

All-day session. New England Ophthalmological Society. See page 498.

12 M. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

1:30 P.M. Radio Program—WEEI. "Bacillary Dysentery."

†2:30-4 P.M. Ward visit, Massachusetts Eye and Ear Infirmary.

†4-5 P.M. Seminar, Pediatric Laboratory, Massachusetts General Hospital.

4:30 P.M. Radio Program—WBZ. "Lumps in the Breast."

*8:15 P.M. Robert Breck Brigham Hospital, Clinical Meeting. 125 Parker Hill Avenue, Boston.

Wednesday, March 20—

5:30 P.M. Tufts Medical Alumni Dinner (business meeting). Dinner at 6:30 P.M. University Club, Boston.

8 P.M. New England Physical Therapy Society. Evans Memorial Auditorium, 82 East Concord Street, Boston.

8:15 P.M. Boston City Hospital Staff Clinical Meeting, Cheever Amphitheatre.

Thursday, March 21—

*12 M. Clinico-Pathological Conference. Massachusetts General Hospital.

†12 M. Clinico-Pathological Conference. Children's Hospital.

*3:30 P.M. Medical Clinic. Dr. Christian. Peter Bent Brigham Hospital.

†4:30 P.M. Surgical Clinic. Peter Bent Brigham Hospital.

Friday, March 22—

5 P.M. Radio Program—WEEI. "The X-Ray in Tuberculosis." "Home Care in Tuberculosis."

Saturday, March 23—

*10-12. Medical Staff Rounds. Dr. Christian. Peter Bent Brigham Hospital.

Sunday, March 24—

4 P.M. Harvard University. (Medical School, Building D, Longwood Avenue, Boston.) Free lecture. "Inheritance." Dr. F. C. Irving.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

March 15—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 499.

March 15—The New England Roentgen Ray Society will meet at the Boston Art Club at 8 P.M.

March 18—Boston Medical History Club, Boston Medical Library, and the Greater Boston Medical Association. See page 499.

March 19—Robert Breck Brigham Hospital, Clinical Meeting. See page 499.

March 19—Lawrence Cancer Clinic. See page 498.

March 19—The South End Medical Club will meet at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, at 12 noon.

March 19—New England Ophthalmological Society. See page 498.

MASSACHUSETTS DIETETIC ASSOCIATION

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

March 20—New England Physical Therapy Society will meet at the Evans Memorial Auditorium, 82 East Concord Street, Boston, at 8 P.M.

March 20—Boston City Hospital Staff Clinical Meeting will be held in the Cheever Amphitheatre at 8:15 P.M.

March 20—Tufts Medical Alumni Dinner. See page 499.

March 21—Clinic at the Peter Bent Brigham Hospital. See page 498.

March 25—New England Heart Association. See page 499.

March 26—Harvard Medical Society. See page 499.

April 12—William Harvey Society. Dr. Jonathan C. Meakins, President American College of Physicians, will speak on "Cardiology During the Past Three Hundred Years—The Legacy of William Harvey."

April 23—The Massachusetts Society for Social Hygiene will meet at the University Club, Boston. For information address Dr. E. Granville Crabtree, 99 Commonwealth Avenue, Boston.

April 25, 26, and 27—The American Association on Mental Deficiency will meet at the Palmer House, Chicago. For information address the Secretary, Dr. Groves B. Smith, Godfrey, Illinois.

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details, address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

June 11—American Heart Association. See page 499.

June 12 and 13—Academy of Physical Medicine. See page 499.

June 17 to 21—Convention of the Catholic Hospital Association will be held at Creighton University, Omaha, Nebraska. For information address the Most Reverend Joseph Francis Rummel, D.D., Bishop of Omaha.

June 27-29 inc.—British National Association for the Prevention of Tuberculosis will be held at Southport, England. Persons desiring further information should write to Miss F. Stickland, Secretary of the Association at Tavistock House North, Tavistock Square, London, W. C. I., England.

July 22-27—Seventh International Congress on Industrial Accidents and Diseases, Brussels, Belgium. The American Committee of the Congress is under the chairmanship of Dr. Fred H. Albee, New York, for the Section on Accidents, and that of Dr. Emery R. Hayhurst, Columbus, Ohio, for Industrial Diseases. The American delegation to the Congress will sail from New York on July 8 and visit London, Amsterdam, The Hague and Paris, and, optionally, Budapest. Physicians interested in the Congress or in the medical tour in conjunction with it, may address the Secretary, Dr. Richard Kovacs, 1100 Park Avenue, New York City.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Annual Meeting will be held in May. Time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meeting will be held on the second Tuesday of May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

May 8—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

March 26—Fernald School for Feeble-Minded, Waverley. Details to be announced.

May—Annual Meeting. Date, time and place to be announced.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March 21—Meeting will be held at the Plymouth County Hospital, South Hanson, at 11 A.M.

April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 27—Clinical Meeting at the Boston Lying-In Hospital.

April 24—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

April 10—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M.: Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.